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Original Communications

PROGRESSIVE CHANGES IN PULMONARY FUNCTION AFTER PNEUMONECTOMY: THE INFLUENCE OF THORACOPLASTY, PNEUMOTHORAX, OLEOTHORAX, AND PLASTIC SPONGE PLOMBAGE ON THE SIDE OF PNEUMONECTOMY

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THE fate of the remaining lung after pneumonectomy has been a matter of great concern to thoracic surgeons and the subject of a number of interesting and revealing physiologic studies.[1-5] As pneumonectomy is being carried out successfully for an increasing variety of diseases including tuberculosis, and as the average age of the patients operated upon decreases, establishment of optimal conditions for the preservation of function of the remaining lung becomes increasingly important.

Previous investigations of lung volume after pneumonectomy have demonstrated that a variable amount of overdistention of the remaining lung occurs, though this is not as severe in man as in the experimental animal.[1, 2, 4, 6, 7] Overdistention is generally thought to be undesirable in patients with pulmonary tuberculosis. The current thought is that a lung with a larger volume than normal doing twice its normal work is more susceptible to reactivation of disease and handles residual disease poorly.[8] Overdistention causes a variety of complaints due to displacement of the mediastinum. The worst of these is ineffective cough due to angulation of the trachea.

Both Birath and Cournand have demonstrated that intrapulmonary mixing of gases is impaired in the *remaining* overdistended lung.[1, 2] The great variability of the physiopathologic pattern was well shown by follow-up studies after pneumonectomy in childhood.[9, 10] Cournand suggested early that thoracoplasty might prevent the development of contralateral pulmonary distention without further impairing maximal function.[1] However, during a recent extensive

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study of cardiorespiratory function after total resection he showed that thoracoplasty on the pneumonectomy side leading to scoliosis reduces the ventilatory effectiveness of the remaining lung.[4]

Investigations of pulmonary function after pneumonectomy have provided only a partial answer to the problem of the management of the empty hemithorax for several reasons. The number of patients studied was usually small. Often it was evident that older patients, in whom emphysema of the remaining lung was found, must have had this disease prior to operation. Following resection for bronchogenic carcinoma it was frequently not clear whether progressive loss of function was due to overdistention or extension of the tumor.[3] Studies performed on one or two occasions some time after pneumonectomy did not reveal during what period after resection the changes in the remaining lung had occurred. Lastly, since the volume and function of the "good" lung alone prior to operation was never known, it has not been clear whether functional changes after pneumonectomy were due to resection of functioning lung tissue, due to progressive physiologic changes of the remaining lung, or due to a combination of both.

For an understanding of the development of functional defects of the remaining lung and for a rational approach toward establishment of ideal conditions for functional preservation of the remaining lung, a plan of investigation was made which was thought necessary for a solution to the problem of overdistention. Prior to operation a bronchospirometric method was used which permitted not only determination of oxygen uptake, ventilation, and vital capacity, but also permitted determination of residual volume, total lung capacity, and pulmonary mixing indices for the two lungs separately.[11] The state of overdistention of the remaining lung could therefore be established prior to operation. The residual volume technique was modified for this study to permit appreciation of small changes in this value.[11] To establish the time after operation when the greatest changes in the remaining lung take place, patients were studied at weekly intervals immediately after operation and at monthly intervals thereafter. To obtain an adequate long-term follow-up of all patients studied prior to operation, patients with carcinoma were excluded from the study.

METHOD

The Darling open circuit oxygen method[12] for determination of the total lung capacity was modified for this investigation, and a new bronchospirometric technique was developed for determination of differential lung volumes. These methods will be described in more detail elsewhere.[11]

Total Lung Capacity.—The final volume of gas obtained by the open circuit method depends on the exact place in the respiratory cycle at which the patient is switched from room air to oxygen breathing. This circuit change is made at the end of a quiet or forced expiration, depending on whether one desires to measure the functional residual or the true residual fractions of the lung volume. Since it is difficult to switch at the very end of forced expiration, particularly in patients who are somewhat dyspneic, the change is usually made at the end of a quiet respiration.[13] The correct timing is ascertained by observing the patient's respiratory movements. In a normal subject, the functional residual volume averages 1,500 cubic centimeters. If a switching error of 150 c.c. or one-fourth of a tidal breath is made, this introduces an error of 10 per cent in the calculated functional residual volume. After pneumonectomy, the functional residual volume averages 800 c.c., and not infrequently 500 c.c. or less. A similar error in estimation of the end of a quiet expiration then causes an error of 20 to 30 per cent of the calculated volume, a much larger variation in itself than the true changes in volume which may be expected to take place from month to month after pneumonectomy. Furthermore, correct timing of the change from room air to oxygen is made more difficult by the more rapid respiratory rate shortly after pneumonectomy. All calculations are based on the assumption that the lungs initially contained 81 per cent nitrogen. Ordinary recording spirometry, therefore, cannot be used prior to lung volume determination, since this would change the initial composition of gas in the lungs.

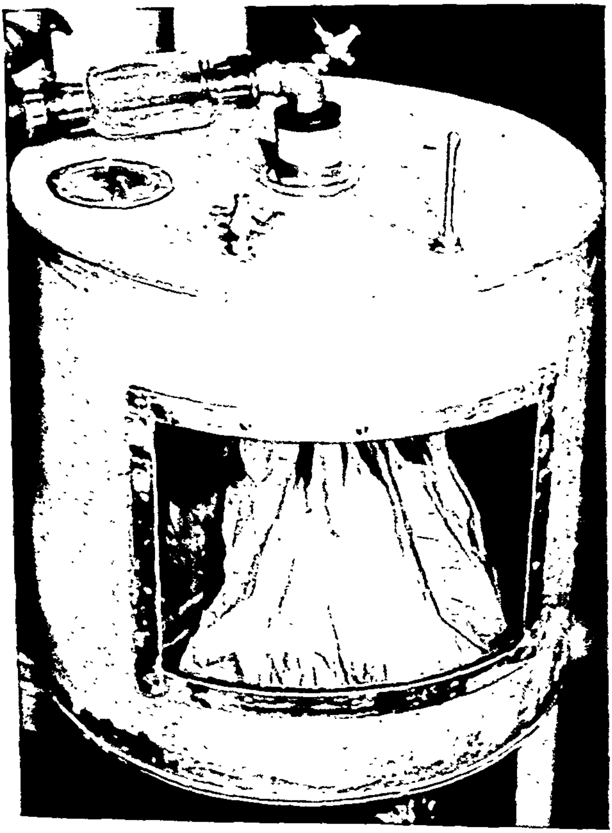

Fig. 1.—Balloon within air-tight box permitting ambient air spirometry.

Three changes were made to increase the accuracy of the method. Prior to oxygen breathing, patients inspired room air from a rubber balloon enclosed in an airtight box and expired into the box around the balloon* (Fig. 1). The box was also connected to a recording spirometer. As the patient inspires, the pressure in the box decreases and the spirometer bell descends and, as the patient expires, the reverse takes place. Thus, although the patient breathes room air, a spirogram can still be obtained. This permits not only better observation of the breathing cycle, but any switching error can later be measured from the spirogram and added or deducted from the calculated functional residual volume (Fig. 2). Two further changes were made. An alveolar trap was used for collection of a terminal alveolar sample as suggested by Wright.[14] This assures collection of alveolar air even in patients who are dyspneic. Finally, an oxygen flushed Douglas bag was used instead of a Tissot spirometer for collection of expired gas. This obviated dead space correction, Tissot rinsing, and errors from changing dead space due to water evaporation and changing spirometer position.

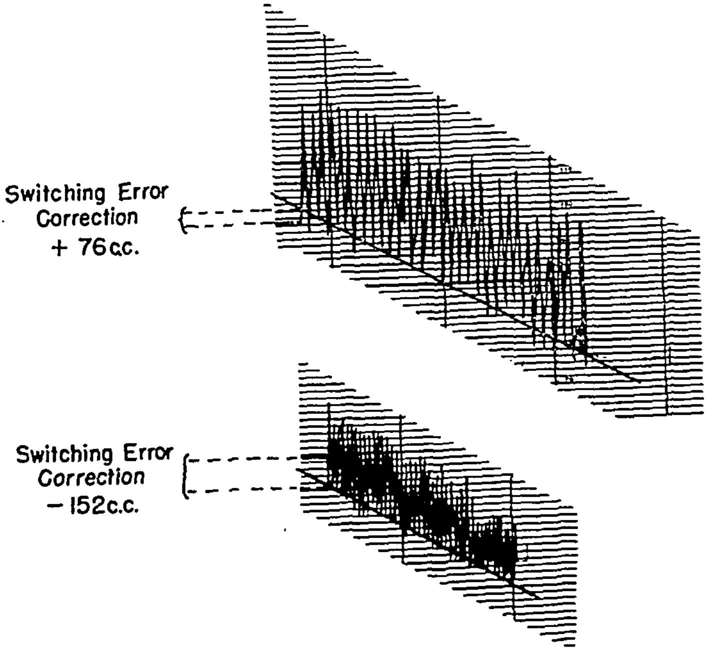

Fig. 2.—Ambient air spirograms obtained prior to oxygen breathing for determination of the functional residual capacity. The switching error can be measured from the tracing.

Ambient Air Bronchospirometry.—Preoperative bronchospirometry was carried out in thirty-one of the forty patients studied. Four patients were not seen until shortly after operation; routine preoperative bronchoscopy revealed an entirely obliterated main stem bronchus in two patients, and three patients had ulcerative endobronchial disease in the left main stem bronchus. Nine patients were therefore excluded.

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*This instrument can be obtained from Warren E. Collins, Inc., 555 Huntington Ave., Boston, Mass.

Recording bronchospirometry with patients breathing room air was made possible by use of a twin spirometer fitted with two balloon-box attachments described above. The connections between boxes and spirometers were made in such a way that either room air or oxygen could be supplied to either lung by the turn of a single valve.* During the course of this and other studies, good evidence was obtained that oxygen spirometry places a greatly damaged lung at a relative advantage. The percentage of the total oxygen uptake of the diseased lung can sometimes be overestimated during oxygen bronchospirometry, and we have therefore preferred ambient air bronchospirometry.[11]

Differential Total Lung Capacity.—Ambient air spirometry made it possible to determine the correct spirometer tube position, oxygen uptake, ventilation, and vital capacity without contaminating the lungs with oxygen. The procedure could therefore be followed immediately by determination of functional residual volume of both lungs separately using one open circuit apparatus for each lung. The efficiency of intrapulmonary mixing could be estimated by collection of alveolar samples of each lung separately at the end of the seven-minute oxygen breathing period.[11] The entire procedure required intubation for twelve to fifteen minutes. Difficulty with this technique was encountered because of changes in the catheter's position during oxygen breathing.[15] During the past year the use of a Carlens catheter[16] with its rubber leg engaging the carina has greatly facilitated this procedure. From the differential residual volume, and the two switching error corrections, it was possible to calculate the two total lung capacities and their subdivisions.

Ventilation Studies.—The maximum breathing capacity (MBC) was obtained using a high velocity respiratory valve and a Douglas bag.[14] The best of three efforts was recorded. The gas was collected for thirty seconds, and the volume was measured in a Tissot spirometer and reported in liters per minute at 37° C.

The exercise test consisted of a level walk at the rate of 180 feet per minute.[17] The expired air was collected during the second to fourth minutes of this exercise. The volume was measured like that of the MBC, and was also reported in liters per minute at 37° C. The walking index was calculated from the equation $\frac{\text{Walking ventilation}}{\text{MBC}} \times 100.$[17]

The best of three upright vital capacity (VC) determinations was recorded. During the past year an attachment for the vital capacity spirometer was used which records not only the total vital capacity but also, and simultaneously, the volume of air expired during the first, the first two, and the first three seconds of the effort.[18]* These timed volumes were reported as per cent of the total vital capacity. Normal volunteers and patients without obstructive ventilatory defect were able to expire on the average 83 per cent of the total vital capacity in the first second, 94 per cent in the first two seconds, and 97 per cent in the first three seconds.[15] The predicted MBC and VC were calculated from Cournand's regression formulas.[13] One-half of the predicted value was used as "normal" for patients after pneumonectomy. The air velocity index was calculated from the equation $\frac{\text{per cent of predicted MBC}}{\text{per cent of predicted VC}}$.[19] This index should be 1.0 or larger in the absence of any obstructive ventilatory defect.[19]

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The supine vital capacity and its subdivisions, minute ventilation, minute oxygen uptake, and respiratory rate were obtained by closed spirometry. The ventilatory equivalent for oxygen was calculated from the equation $\frac{\text{minute ventilation in liters.}}{\text{minute oxygen uptake in c.c.}}$. It was reported as number of liters ventilated per 100 c.c. oxygen removed.

Distributional and Diffusional Factors.—A rough measure of the adequacy of intrapulmonary mixing was obtained from the nitrogen content of the alveolar sample after seven minutes' oxygen breathing.[12] In normal subjects this sample contains no more than 2.5 per cent nitrogen. Cournand and associates recently observed that this value is even lower after pneumonectomy.[4] Moderate hyperventilation during the oxygen breathing period and a greatly reduced dead space and functional residual volume probably accounts for this. From data of the present investigation it was concluded that in patients after pneumonectomy an alveolar nitrogen content of over 1.5 per cent should be considered indicative of defective intrapulmonary mixing.

Early in the study, arterial samples were obtained and analyzed for oxygen and carbon dioxide content and oxygen combining power, and the arterial oxygen saturation was calculated. These studies were abandoned after a few months, chiefly because all resting oxygen saturations were well within normal limits. A large number of patients of this group were operated upon for tuberculosis, and it was felt that exercise severe enough to demonstrate arterial unsaturation was contraindicated. Finally a number of patients who gladly volunteered for ventilation studies hesitated to return to the laboratory for weekly or monthly arterial punctures.

Nomenclature.—The new definitions recently proposed by a committee of respiratory physiologists were adopted because they are simple and self-explanatory.[20] The subdivisions of the *total lung capacity* were (1) the *tidal volume* moved during resting ventilation, (2) the *inspiratory capacity* or maximal volume of inspired air measured from the pulmonary mid position, (3) the *expiratory reserve* or maximal volume of expired air measured from the pulmonary midposition, (4) the *residual volume* remaining in the lungs after maximal expiration, and (5) the *functional residual capacity* obtained by addition of the expiratory reserve and residual volume.

CLINICAL MATERIAL AND STUDY PROGRAM

Patients were selected in whom pneumonectomy was planned for disease other than primary or metastatic malignant tumor. Operation was performed for tuberculosis in thirty-five patients, for multiple lung abscesses in two, for total bronchiectasis in one, and for myoblastoma in one. Pneumonectomy and pleurectomy were performed in one patient for total mixed empyema with multiple bronchopleural fistulas.

Nine patients had additional collapse of the remaining lung. Pneumoperitoneum was induced prior to operation and maintained throughout the period of study in eight patients. One patient had a five-rib thoracoplasty over the remaining lung prior to pneumonectomy. The functional effects of the pneumoperitoneum were ignored in the present statistical analyses because we were chiefly concerned with changes in function and the pneumoperitoneum remained unchanged both before and after operation. Furthermore, the changes due to pneumoperitoneum in patients with pneumonectomy are extremely small.[5]

No patient had significant disease from a functional standpoint of the remaining lung either before or after operation. Spreads in this series, when they occurred immediately after surgery, invariably disappeared within two to four months and were thought to have no measurable effect on pulmonary ventilation.

All but four patients were studied immediately prior to operation and all patients were studied one and two weeks, and two, three, four, and six months after operation, and every six months thereafter. Only patients studied at least six months after operation were included in the study. The walking test was omitted for the first four months after operation in all patients with tuberculosis. The MBC and upright VC tests were omitted at the one week postoperative study. One patient, after having been well for two years, suddenly developed a tuberculous abscess of the chest wall and died, presumably of miliary tuberculosis, shortly thereafter. All of the remaining thirty-nine patients are essentially well six months to two and one-half years after operation.

TREATMENT OF DATA

Each time a patient was studied, a total of twenty-two determined and derived values were obtained, excluding the preoperative bronchospirometry and differential residual volume determination. The forty patients were studied on 343 occasions, and 8,918 data were obtained. It was not possible to show these either in tabular form or in illustrations. The data were therefore presented in two ways. Typical patients' data are shown for every one of the various methods of treatment of the pneumonectomy side. In addition, statistical data are presented in summary form for each method of treatment. The number of patients in some groups was small, and still fewer patients were studied at the longer time intervals. The summary illustrations were thereby limited to one and one-half years after operation.

1. *Pneumonectomy Without Aftertreatment.*—Twelve patients received no further treatment of the empty hemithorax after resection except for two or three thoracenteses during the first postoperative week for pressure adjustment and removal of excess fluid. Penicillin and streptomycin were injected daily for two weeks.

CASE 1.—*Little Postoperative Overdistention.*—M. B., a 27-year-old woman, was operated on for tuberculosis with a completely destroyed lung (Fig. 3, Table I).

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Bronchospirometry showed no ventilation or gas exchange by the left lung. Both the VC and the MBC were larger than 50 per cent of predicted, and the air velocity index was 1.37. The residual volume and total capacity were also larger than the predicted normal for one lung. The lung was therefore somewhat hyperinflated during all phases of respiration. Residual volume expressed as per cent of total capacity (RV/TC x 100) was 26 per cent or essentially within normal limits. The pulmonary mixing index of 4.67 was greatly elevated, probably because the atelectatic and excavated left lung was not ventilated but did have nitrogen containing dead space.

Immediately after operation all components of the total capacity were decreased. The expiratory reserve suffered most, due to hesitation by the patient to make a painful forced expiration. The residual volume was reduced more than the total capacity and the RV/TC ratio was decreased to slightly below the predicted figure. These immediate postoperative findings were characteristic for all patients after primary pneumonectomy. The total capacity was reduced both because pain inhibited maximal inspiration and because the lung was somewhat compressed by large quantities of fluid and air under neutral pressure in the empty hemithorax. The respiratory rate, the minute ventilation, the ventilatory equivalent for oxygen, and the minute oxygen uptake were all increased. As the tidal volume decreased, respiration became less efficient because the dead space occupied a relatively larger portion of each breath. This patient breathed 3.7 liters for every 100 c.c. of oxygen removed, whereas only 3.0 liters of air were required before operation.

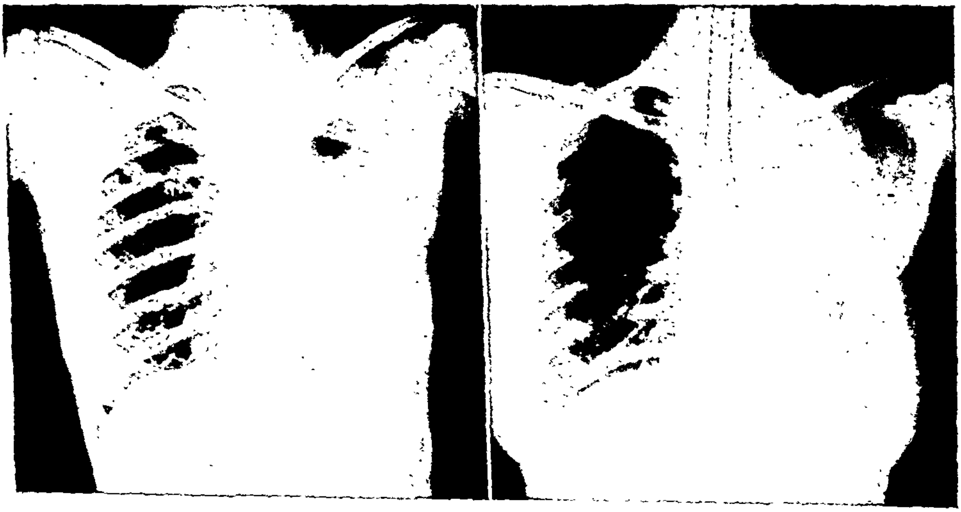

Fig. 3.—Roentgenograms of the chest of patient M. B. before operation and two years after pneumonectomy. Little overdistention was noted without further treatment.

During the following two months the total capacity returned to its preoperative level, and the vital capacity remained reduced by about 100 c.c. at the expense of the residual volume which increased about 100 c.c. The lung at full inspiration was no more overdistended than before operation (Fig. 3), but the resting or midposition at the end of a quiet expiration was somewhat higher. This increase of the RV/TC ratio to 30 per cent is sometimes considered to represent slight emphysema.[21, 22]

During the next one and one-half years no further striking changes occurred. The oxygen uptake increased as the patient gained 45 pounds, and minute ventilation and the ventilatory equivalent returned to the preoperative values. The pulmonary mixing index was sharply reduced immediately after operation, and never increased above 1.76 per cent nitrogen after that.

TABLE I. PNEUMONECTOMY WITHOUT AFTERTREATMENT, LITTLE OVERDISTENTION (PATIENT M. B.)

	PREOPERATIVE					POSTOPERATIVE							
FUNCTION TEST	BRON. SPIR. RIGHT	TOTAL	ONE LUNG PRED.	LEFT	RIGHT	1 WK. RIGHT	2 WK. RIGHT	2 MO. RIGHT	3 MO. RIGHT	4 MO. RIGHT	6 MO. RIGHT	1 YR. RIGHT	1½ YR. RIGHT
MBC, L./min.	--	66	43	0	66	--	53	63	64	61	60	57	59
Upright VC., c.c.	--	1,720	1,525	0	1,720	--	1,330	1,700	1,730	1,700	1,760	1,690	1,680
Air velocity index	--	1.37	1.0	-	1.37	--	1.41	1.30	1.30	1.27	1.21	1.20	1.34
Walking ventil., L./min.	--	20	15.2	0	20	--	--	--	--	--	20	20	20
Walking index, %	--	30	36	-	30	--	38	32	31	33	33	35	40
Basal resp. rate	19	19	--	-	19	24	21	20	20	20	20	18	19
Tidal volume, c.c.	100%	380	--	0	380	380	420	380	365	370	375	400	405
Inspir. capacity, c.c.	100%	1,350	--	0	1,350	1,190	1,190	1,280	1,280	1,300	1,180	1,250	1,350
Expiratory reserve, c.c.	100%	240	--	0	240	90	110	200	180	200	230	220	240
Vital capacity, c.c.	100%	1,590	1,525	0	1,590	1,280	1,300	1,480	1,460	1,500	1,410	1,470	1,590
Residual volume, c.c.	100%	560	380	0	560	300	410	630	600	670	660	720	740
Total lung capacity, c.c.	100%	2,150	1,905	0	2,150	1,580	1,710	2,110	2,060	2,170	2,070	2,190	2,330
RV/TC × 100, %	26	26	20	-	26	19	24	30	29	31	32	33	32
Ventilation, L/min.	100%	7,180	--	0	7,180	9,160	8,880	7,620	7,300	7,460	7,520	7,210	7,620
Oxygen uptake, c.c./min.	100%	240	--	0	242	251	232	219	226	230	243	236	277
Ventil. equiv. L./100 c.c.	3.0	3.0	2.2	0	3.0	3.7	3.8	3.5	3.2	3.2	3.1	3.1	2.8
Mixing index, %N_2	4.67	4.67	--	-	4.67	0.63	0.88	0.62	0.81	1.76	1.11	0.92	0.65

Bronchospirometry showed no ventilation or gas exchange by the left lung. Both the VC and the MBC were larger than 50 per cent of predicted, and the air velocity index was 1.37. The residual volume and total capacity were also larger than the predicted normal for one lung. The lung was therefore somewhat hyperinflated during all phases of respiration. Residual volume expressed as per cent of total capacity (RV/TC × 100) was 26 per cent or essentially within normal limits. The pulmonary mixing index of 4.67 was greatly elevated, probably because the atelectatic and excavated left lung was not ventilated but did have nitrogen containing dead space.

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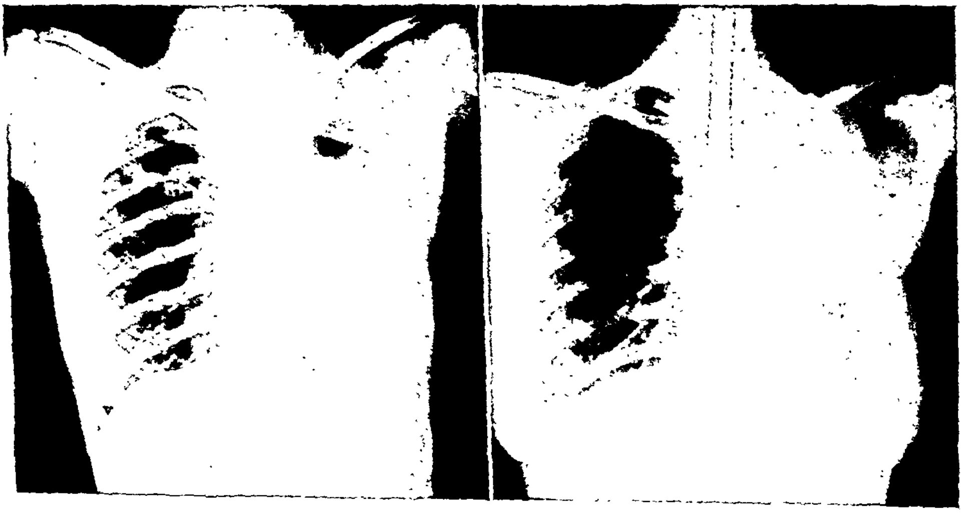

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During the next one and one-half years no further striking changes occurred. The oxygen uptake increased as the patient gained 45 pounds, and minute ventilation and the ventilatory equivalent returned to the preoperative values. The pulmonary mixing index was sharply reduced immediately after operation, and never increased above 1.76 per cent nitrogen after that.

TABLE II. PNEUMONECTOMY FOLLOWED BY MARKED OVERDISTENTION AND EARLY THORACOPLASTY (PATIENT R. M.)

	PREOPERATIVE					AFTER PNEUMONECTOMY				AFTER THORACOPLASTY			
FUNCTION TEST	BRON. SPIR. LEFT	TOTAL	ONE LUNG PRED.	RIGHT	LEFT	1 WK. LEFT	2 WK. LEFT	2 MO. LEFT	3 MO. LEFT	2 WK. LEFT	2 MO. LEFT	4 MO. LEFT	1 YR. LEFT
MBC, L./min.	--	52	48	--	--	--	41	43	43	38	39	31	36
Upright VC, c.c.	--	1,900	1,540	--	--	--	1,560	1,560	1,550	1,250	1,350	1,300	1,590
Air velocity index	--	0.87	1.0	--	--	--	0.84	0.90	0.90	0.98	0.93	0.76	0.73
Walking ventil., L./min.	--	12	16	--	--	--	--	--	--	--	--	.15	.14
Walking index %	--	23	34	--	--	--	29	28	28	31	32	49	38
Basal resp. rate	15	15	--	15	15	22	18	14	14	18	14	15	15
Tidal volume, c.c.	78%	360	--	80	280	330	365	420	400	340	380	340	370
Inspir. capacity, c.c.	86%	1,600	--	220	1,380	1,210	1,400	1,370	1,350	1,160	1,160	1,155	1,250
Expiratory reserve, c.c.	100%	100	--	0	100	0	90	105	100	100	120	125	105
Vital capacity, c.c.	87%	1,700	1,540	220	1,480	1,210	1,490	1,475	1,450	1,260	1,280	1,280	1,355
Residual volume, c.c.	66%	595	385	200	395	270	370	725	760	390	400	380	435
Total lung capacity, c.c.	82%	2,295	1,925	420	1,875	1,480	1,860	2,200	2,210	1,650	1,680	1,660	1,790
RA/TC × 100, %	21	26	20	48	21	18	20	33	34	24	24	23	24
Ventilation, L./min.	78%	5,420	--	1,210	4,210	7,300	6,550	5,880	5,600	6,080	5,320	5,090	5,530
Oxygen uptake, c.c./min.	89%	226	--	25	201	261	234	228	208	230	205	215	254
Ventil. equiv. L./100 c.c.	2.1	2.4	2.2	4.8	2.1	2.8	2.8	2.6	2.7	2.6	2.6	2.4	2.2
Mixing index, % N_2	1.06	2.12	--	5.71	1.06	0.52	0.34	1.06	1.26	0.68	0.38	0.92	0.67

Studies of this patient illustrate that (1) overdistention occurs chiefly during the first two months after operation (2) overdistention postoperatively remains minimal in certain patients, and (3) pneumonectomy for a functionless lung causes no further loss of maximal function as expressed by MBC and VC.

CASE 2.—*Early Severe Postoperative Overdistention:* R. M., a 24-year-old woman, had a pneumonectomy for giant tension cavity which developed during induction of an unsuccessful pneumothorax for far advanced cavernous pulmonary tuberculosis (Fig. 4, Table II).

Preoperative bronchospirometry and differential lung volume determination showed that oxygen uptake of the right lung was reduced to 11 per cent of the total. Ventilation and vital capacity were almost equally reduced. The residual volume on the right was only slightly reduced and the RV/TC ratio was 48 per cent. This does not indicate emphysema, but merely shows that little of the air within the lung could be expired on maximal effort. The mixing index of 5.71 per cent on that side was greatly elevated. The "good" lung showed a normal relationship of the various components of the total capacity, although all components were somewhat less than 50 per cent of predicted. This patient, unlike the last one, had a slightly smaller than normal lung, perhaps because of the pneumoperitoneum and previous cavernous disease on that side.

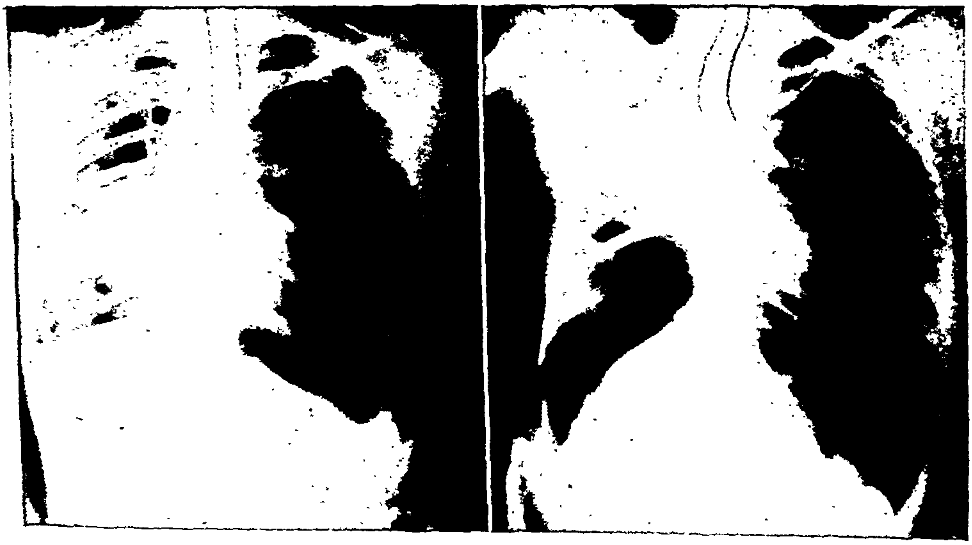

Fig. 4.—Roentgenograms of the chest of patient R. M. before and three months after pneumonectomy. Severe overdistention occurred soon after operation.

Immediately after operation this patient's studies duplicated those of the last patient (M.B.). All components of the total capacity were reduced. As in the last case, ventilation became more inefficient due to increased respiratory rate and decreased tidal volume.

Beginning the second week after operation, the studies of this patient began to show great differences from the previous case. The vital capacity showed no further improvement, but the total capacity increased steadily due to a rising residual volume. At the end of two months, the residual volume was 130 c.c. larger than the combined residual volume before operation and 330 c.c. larger than the residual volume of the "good" lung alone. The RV/TC ratio had nearly doubled (Table II, Fig. 4). Since the right lung participated to some extent in ventilation, its removal, unlike in the last patient, caused a permanent reduction of VC and MBC. Because of these findings, an early thoracoplasty was performed. Changes following this procedure will be discussed later.

TABLE II. PNEUMONECTOMY FOLLOWED BY MARKED OVERDISTENTION AND EARLY THORACOPLASTY (PATIENT R. M.)

FUNCTION TEST	PREOPERATIVE					AFTER PNEUMONECTOMY				AFTER THORACOPLASTY			
	BRON. SPIR. LEFT	TOTAL	ONE LUNG PRED.	RIGHT	LEFT	1 WK. LEFT	2 WK. LEFT	2 MO. LEFT	3 MO. LEFT	2 WK. LEFT	2 MO. LEFT	4 MO. LEFT	1 YR. LEFT
MBC, L./min.	--	52	48	--	--	--	41	43	43	38	39	31	36
Upright VC, c.c.	--	1,900	1,540	--	--	--	1,560	1,560	1,550	1,250	1,350	1,300	1,590
Air velocity index	--	0.87	1.0	--	--	--	0.84	0.90	0.90	0.98	0.93	0.76	0.73
Walking ventil., L./min.	--	12	16	--	--	--	--	--	--	--	--	.15	.14
Walking index %	--	23	34	--	--	--	29	28	28	31	32	49	38
Basal resp. rate	15	15	--	15	15	22	18	14	14	18	14	15	15
Tidal volume, c.c.	78%	360	--	80	280	330	365	420	400	340	380	340	370
Inspir. capacity, c.c.	86%	1,600	--	220	1,380	1,210	1,400	1,370	1,350	1,160	1,160	1,155	1,250
Expiratory reserve, c.c.	100%	100	--	0	100	0	90	105	100	100	120	125	105
Vital capacity, c.c.	87%	1,700	1,540	220	1,480	1,210	1,490	1,475	1,450	1,260	1,280	1,280	1,355
Residual volume, c.c.	66%	595	385	200	395	270	370	725	760	390	400	380	435
Total lung capacity, c.c.	82%	2,295	1,925	420	1,875	1,480	1,860	2,200	2,210	1,650	1,680	1,660	1,790
RA/TC × 100, %	21	26	20	48	21	18	20	33	34	24	24	23	24
Ventilation, L./min.	78%	5,420	--	1,210	4,210	7,300	6,550	5,880	5,600	6,080	5,320	5,090	5,530
Oxygen uptake, c.c./min.	89%	226	--	25	201	261	234	228	208	230	205	215	254
Ventil. equiv. L./100 c.c.	2.1	2.4	2.2	4.8	2.1	2.8	2.8	2.6	2.7	2.6	2.6	2.4	2.2
Mixing index, % N_2	1.06	2.12	--	5.71	1.06	0.52	0.34	1.06	1.26	0.68	0.38	0.92	0.67

Studies of this patient illustrate that (1) overdistention occurs chiefly during the first two months after operation (2) overdistention postoperatively remains minimal in certain patients, and (3) pneumonectomy for a functionless lung causes no further loss of maximal function as expressed by MBC and VC.

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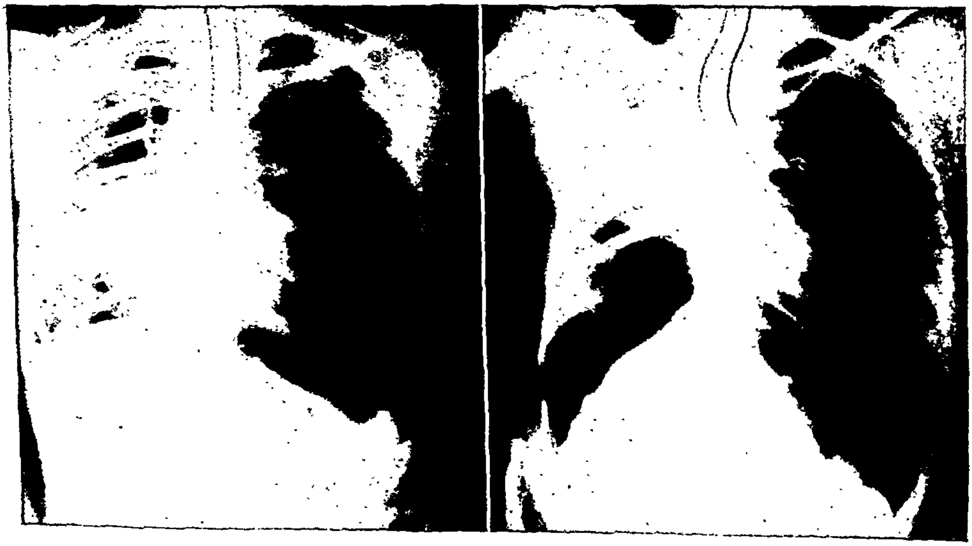

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Immediately after operation this patient's studies duplicated those of the last patient (M.B.). All components of the total capacity were reduced. As in the last case, ventilation became more inefficient due to increased respiratory rate and decreased tidal volume.

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TABLE III. PNEUMONECTOMY WITHOUT AFTERTREATMENT, PREOPERATIVE OVERDISTENTION (PATIENT W. H.)

FUNCTION TEST	PREOPERATIVE					POSTOPERATIVE							
	BRON. SPIR. LEFT	TOTAL	ONE LUNG PRED.	RIGHT	LEFT	2 WK. LEFT	2 MO. LEFT	3 MO. LEFT	4 MO. LEFT	6 MO. LEFT	1 YR. LEFT	1½ YR. LEFT	2 YR. LEFT
MBC, L./min.	--	56	47	--	--	40	42	49	49	50	49	45	44
Upright VC, c.c.	--	2,410	1,840	--	--	1,720	1,900	2,020	2,030	2,060	2,030	2,030	2,030
Air velocity index	--	0.92	1.0	--	--	0.92	0.81	0.95	0.95	0.95	0.95	0.87	0.84
Walking ventil., L./min.	--	25	15	--	--	--	--	--	--	--	13	18	--
Walking index %	--	45	32	--	--	62	60	57	57	50	27	39	40
Basal resp. rate	17	17	--	17	17	--	23	19	18	18	17	21	20
Tidal volume, c.c.	77%	375	--	85	290	--	370	390	350	340	365	460	340
Inspir. capacity, c.c.	93%	1,070	--	75	995	--	790	840	900	860	840	830	975
Expiratory reserve, c.c.	87%	1,130	--	145	985	--	930	960	920	860	1,020	1,020	905
Vital capacity, c.c.	90%	2,200	1,840	220	1,980	--	1,720	1,800	1,820	1,720	1,860	1,850	1,880
Residual volume, c.c.	87%	1,570	565	205	1,365	--	1,140	1,150	1,320	1,190	1,400	1,420	1,085
Total lung capacity, c.c.	89%	3,770	2,405	425	3,345	--	2,860	2,950	3,140	2,910	3,260	3,270	2,965
RV/TC x 100, %	41	42	23.4	48	41	--	40	39	42	41	43	43	37
Ventilation, L./min.	77%	6,360	--	1,460	4,900	--	8,675	7,400	6,350	6,100	6,180	9,640	6,830
Oxygen uptake, c.c./min.	96%	201	--	7	194	--	228	231	205	218	216	290	222
Ventil. equiv. L./100 c.c.	2.5	3.2	2.2	20.8	2.5	--	3.8	3.2	3.1	2.8	2.9	3.3	3.1
Mixing index, % N_2	--	--	--	--	--	--	3.10	2.56	1.98	3.46	3.64	3.73	4.32

This study illustrates that (1) overdistention occurs early after operation, (2) overdistention can be rapid and severe in certain patients, and (3) removal of a lung participating in ventilation to any extent causes a permanent loss of maximal ventilatory function.

CASE 3.—*Overdistention and Emphysema Before Operation:* W. H., a 42-year-old Negro man, had been treated for pulmonary tuberculosis for four years prior to operation. He had a manic-depressive episode immediately after operation, and the one- and two-week studies could not be performed.

Prior to operation, bronchospirometry revealed an almost entirely destroyed lung on the right (Fig. 5, Table III). Differential residual volume determination showed only 11 per cent of the total capacity on the right, and almost one-half of this was residual volume. The "good" lung before operation was greatly overdistended. It alone occupied a space (3,345 c.c.) which was over two-thirds of the total predicted volume (4,810 c.c.). The single vital capacity was just 50 per cent of the total predicted, but the residual volume was nearly twice normal. The RV/TC ratio of the "good" lung alone was increased to 41 per cent. In this patient, this represented true emphysema in part. The timed capacities during the first, the first two, and the first three seconds obtained from the spirogram were only 58, 68, and 77 per cent of the total vital capacity, respectively, showing a definite slowing of the air stream. Further evidence of an obstructive ventilatory defect was given by the air velocity index of less than 1.0. This index is usually higher than 1.0 in patients with chronic loss of ventilating lung tissue without obstructive elements.[19]

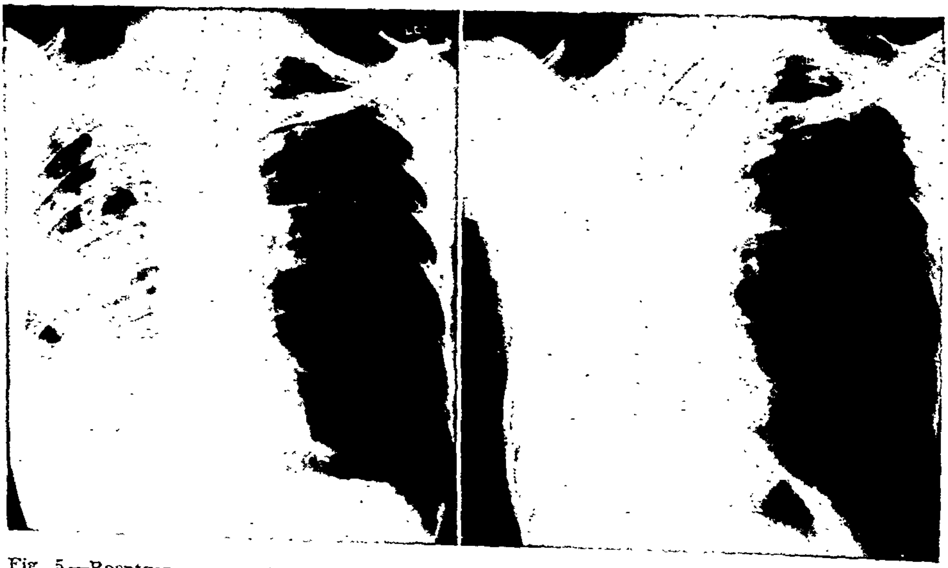


Fig. 5.—Roentgenograms of the chest of patient W. H. before and three years after pneumonectomy. The overdistention prior to resection was marked.

Comparison of the total capacity of the single "good" lung before operation with that during the two years after operation shows essentially no change. No further overdistention occurred. This patient's studies are unusual in three respects. Unlike all other patients, this subject's inspiratory capacity was smaller than his expiratory reserve which was relatively huge. The functional residual volume was therefore greatly elevated. The patient breathed with a greatly overdistended lung which is only partly expressed by the RV/TC ratios of 37 to 43 per cent. The mixing index was much larger than the normal 1.5 per cent nitrogen after pneumonectomy. This patient was also unusual in that he showed evidence of true emphysema which increased during the second year of study. The MBC showed a progressive decline, the air velocity index and breathing reserve during exercise decreased also, and the timed capacities declined while the total vital capacity remained unchanged.

In summary, this patient showed that (1) postoperative overdistention need not necessarily be the result of the removal of the lung, but may have been present to an equal extent prior to operation and (2) that simple overdistention may, at an early date, develop into true obstructive emphysema.

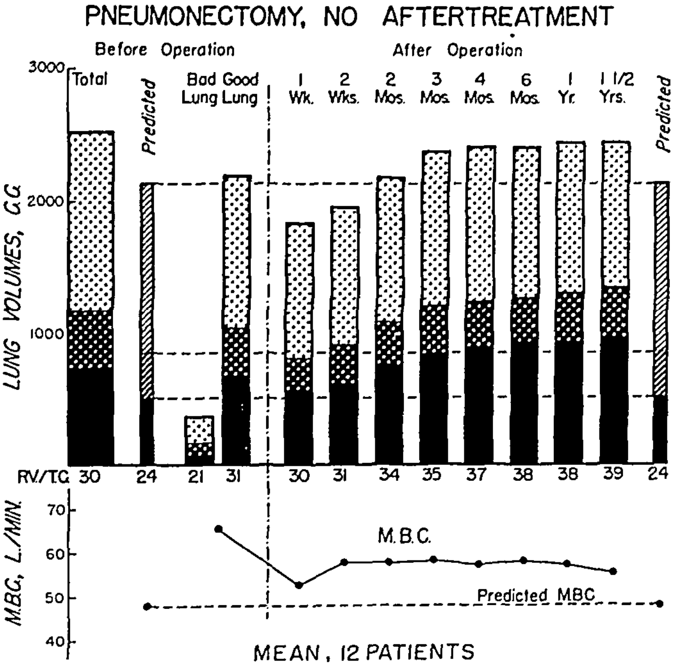


Fig. 6.—Mean lung volumes and maximum breathing capacities of twelve patients not further treated after pneumonectomy. The total lung capacities and subdivisions of both lungs together and each lung separately are shown before operation and at intervals thereafter. The predicted lung volume and maximum breathing capacity for a single lung are indicated. The subdivisions of the total lung capacity in this and subsequent drawings are identified as follows: black: residual volume, stippled black: expiratory reserve, stippled white: inspiratory capacity. The two dark areas together represent the functional residual capacity and the two stippled areas together represent the vital capacity.

Fig. 6 shows a summary of studies of twelve patients who had no further treatment after pneumonectomy. Before operation the average "good" lung showed a normal total capacity, a somewhat reduced vital capacity, and a slightly increased residual volume. Therefore, slight overdistention was present during quiet respiration, but not at fullest inspiration. When compared to the single "good" lung before operation, the inspiratory capacity and expiratory reserve, together the vital capacity, showed a sharp decrease shortly after operation and returned to their preoperative values within two months. The total capacity showed a slight decrease immediately after operation but then increased during the first few months. This increase was entirely due to a sharp increase of the residual volume from 681 c.c. before operation to 903 c.c. four months after pneumonectomy. The RV/TC ratio increased from 31 to 37 per cent. Thus, after operation the average lung was somewhat overdistended both in maximal inspiration and in the pulmonary midposition, more so in the latter.

2. *Pneumothorax.*—In six patients, the fluid was removed from the space formerly occupied by the lung and replaced with air. These patients were selected because their condition was so poor prior to pneumonectomy that it was thought unlikely that they would soon thereafter be able to tolerate further surgery. The space was dried by thoracentesis immediately after operation and every day thereafter for fourteen days. Thoracenteses were followed by streptomycin and penicillin injections. Usually no further punctures were necessary after this time. Pressures were maintained slightly on the negative side for two weeks. The amount of weekly refills was then governed by the position of the mediastinum seen by fluoroscopy and by reports of lung volumes from the laboratory. Neutral mean pressures usually sufficed for the first month, and mean pressures of plus 3 and plus 5 cm. of water were needed thereafter. In one patient, reappearance of clear yellow fluid four months after operation was indicative of tuberculous empyema. This was first treated by lavage and later by oleothorax.

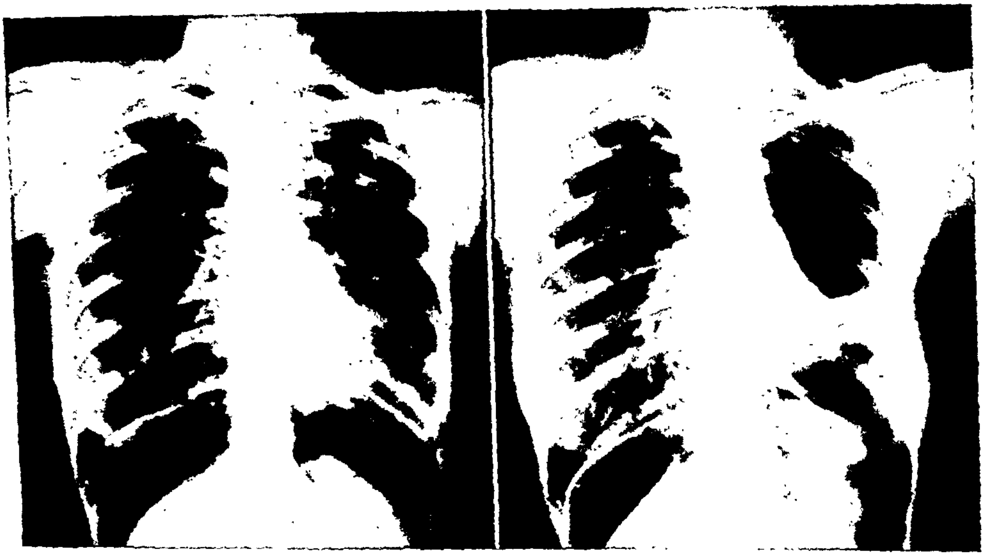


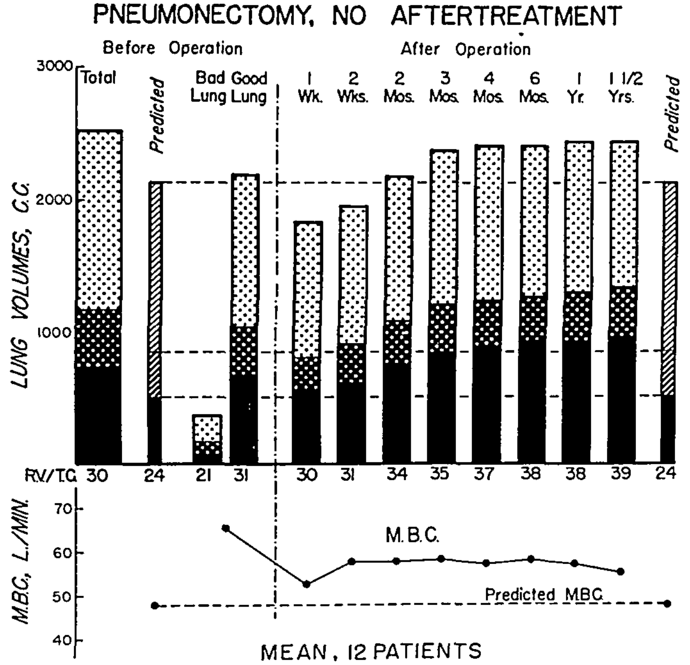
Fig. 7.—Roentgenograms of the chest of patient A. L. before pneumonectomy and six months after pneumothorax treatment of the space. No overdistention was observed.

CASE 4.—A. L., a 23-year-old woman, had a pneumonectomy for multiple tuberculous excavations in both lobes of the left lung (Fig. 7). The patient was very ill and toxic, and weighed only eighty-six pounds.

Bronchospirometry prior to operation showed that the left lung contributed about one-fourth of the total ventilation, oxygen uptake and vital capacity; differential residual volume determination showed 35 per cent of the total residual volume on the left (Table IV). In spite of the advanced disease, the MBC was 108 per cent of predicted for two lungs, and the upright VC was 100 per cent of predicted. The "good" right lung was much larger than expected. Its total capacity was 136 per cent of the calculated normal volume. However, the components of this total capacity had a normal relationship, and the RV/TC ratio was 20 per cent (normal).

normal 1.5 per cent nitrogen after pneumonectomy. This patient was also unusual in that he showed evidence of true emphysema which increased during the second year of study. The MBC showed a progressive decline, the air velocity index and breathing reserve during exercise decreased also, and the timed capacities declined while the total vital capacity remained unchanged.

In summary, this patient showed that (1) postoperative overdistention need not necessarily be the result of the removal of the lung, but may have been present to an equal extent prior to operation and (2) that simple overdistention may, at an early date, develop into true obstructive emphysema.



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Immediately after operation, the changes were the same as in the previous cases. All components of the total capacity were reduced, the residual volume more so than the others, probably due to high pneumothorax pressures.

During the first postoperative year, an entirely different picture developed than in the patients without treatment. At the end of three months, all components of the total capacity had regained the values of the single "good" lung before operation, and the normal relationship between vital capacity and residual volume persisted. This RV/TC ratio remained normal throughout the period of study. The preoperative residual volume

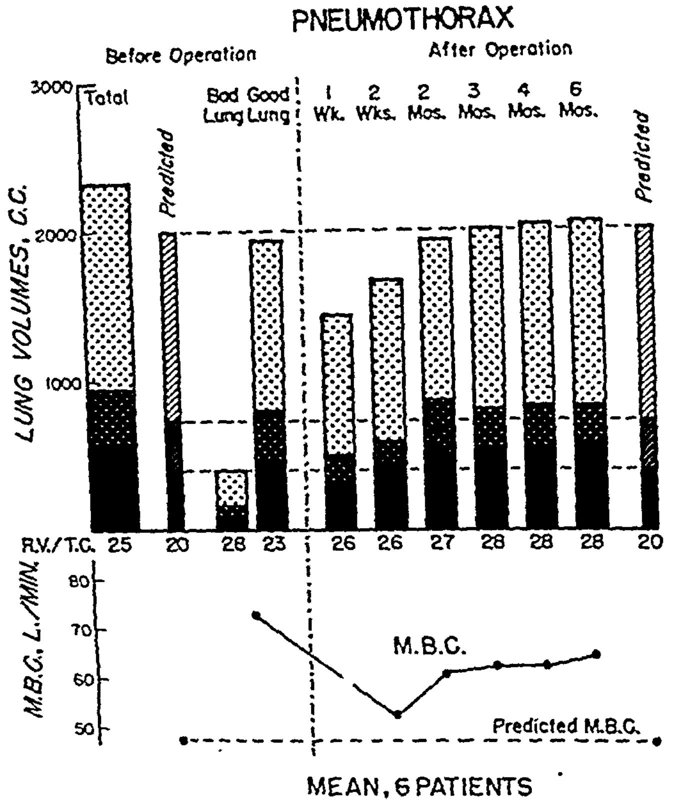

Fig. 8.—Mean function studies of 6 patients in whom pneumonectomy was followed by pneumothorax.

of the single lung was 490 c.c., and this same volume was 510 c.c. one year later. Maximal preservation of function of the remaining lung was further shown by a maximum breathing capacity, which was 182 per cent of the normal predicted for one lung and by a vital capacity of 152 per cent of predicted. Although this patient's lung was much larger than predicted, it also functioned much better in every respect. Since the RV/TC ratio remained normal, this large size could not be regarded as evidence of overdistention, but rather as the remaining lung of a patient with unusually good pulmonary function prior

Fig. 8 shows a summary of the postoperative studies of six patients maintained on pneumothorax. The immediate postoperative changes were the same as in the previous chart. At the end of three months there was no evidence of overdistention and, although the residual volume slowly increased, at the end of one year it was only 80 c.c. larger than the calculated single residual volume before operation. The RV/TC ratio increased only 4 per cent. The MBC was

TABLE IV. PNEUMONECTOMY FOLLOWED BY PNEUMOTHORAX (PATIENT A. L.)

FUNCTION TEST	PREOPERATIVE					POSTOPERATIVE						
	BRON. SPIR. RIGHT	TOTAL	ONE LUNG PRED.	LEFT	RIGHT	1 WK. RIGHT	2 WK. RIGHT	2 MO. RIGHT	3 MO. RIGHT	4 MO. RIGHT	6 MO. RIGHT	1 YR. RIGHT
MBC, L./min.	--	98	46	--	--	--	70	79	81	83	82	83
Upright VC, c.c.	--	2,910	1,465	--	--	--	2,260	2,240	2,240	2,260	2,270	2,230
Air velocity index	--	1.09	1.00	--	--	--	1.00	1.16	1.19	1.20	1.18	1.20
Walking ventil., L./min.	--	17	15.2	--	--	--	--	--	--	--	17	17
Walking index, %	--	17	34	--	--	--	24	22	22	20	21	21
Basal resp. rate	19	19	--	19	19	23	19	17	17	17	16	16
Tidal volume, c.c.	--	340	--	80	260	365	405	345	335	355	325	375
Inspir. capacity, c.c.	67%	1,950	--	635	1,315	1,270	1,410	1,400	1,450	1,440	1,410	1,475
Expiratory reserve, c.c.	100%	690	--	0	690	260	300	420	460	450	440	525
Vital capacity, c.c.	76%	2,640	1,465	635	2,005	1,530	1,710	1,820	1,910	1,890	1,850	2,000
Residual volume, c.c.	65%	750	365	260	730	310	370	450	450	470	490	510
Total lung capacity, c.c.	74%	3,390	1,830	895	2,495	1,840	2,080	2,270	2,360	2,360	2,340	2,510
RV/TC x 100, %	20	22	20	29	20	17	18	20	19	20	21	20
Ventilation, L./min.	76%	6,500	--	1,700	4,800	8,440	7,570	5,880	5,700	6,030	5,240	6,020
Oxygen uptake, c.c./min.	80%	224	--	45	179	248	244	226	228	232	218	242
Ventil. equiv. L./100 c.c.	2.7	2.9	2.2	3.8	2.7	3.4	3.1	2.6	2.5	2.6	2.4	2.5
Mixing index, % N_2	--	1.84	--	1.06	2.27	0.62	0.81	0.76	0.94	0.47	0.92	0.71

138 per cent of the "one lung" predicted, and the VC was 94 per cent of this same value.

Pneumothorax completely confined the remaining lung within its normal position and volume, and it did so without causing any further reduction of maximal ventilation.

3. *Oleothorax.*—Two patients were maintained on pneumothorax for four and nine months, respectively, and the space was then filled with oil. This was done in one patient because of the appearance of clear yellow fluid containing *M. tuberculosis*, and in the other, because of appearance of sterile cloudy fluid. A third patient, not included in this series, has been followed for three years with oleothorax on one side and an extrapleural pneumothorax of the remaining lung.[5] The oil in the first two cases was mineral oil, and in the last case, olive oil. Formation of fluid subsided in the first two patients shortly after introduction of the oil, and all three patients are well at present.

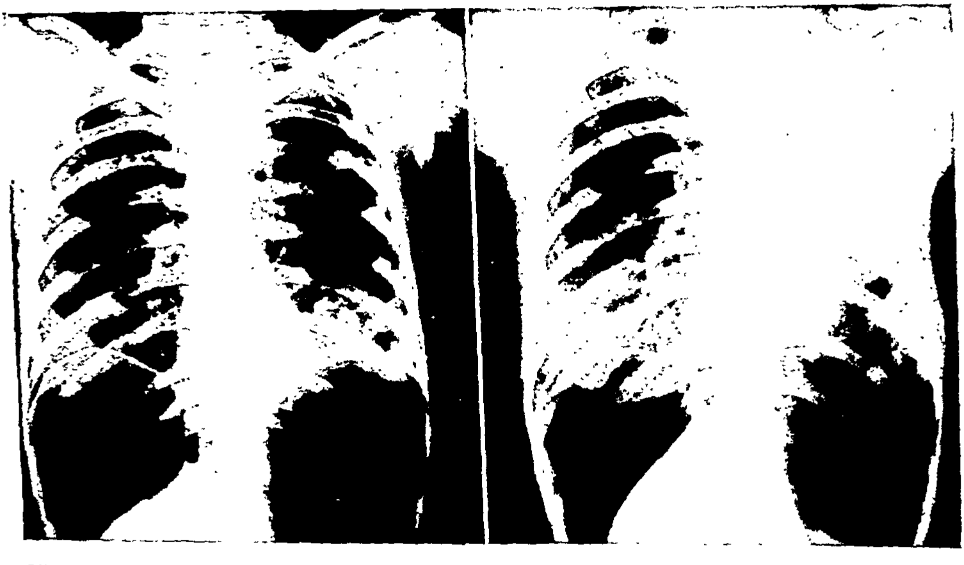

Fig. 9.—Roentgenograms of the chest of patient D. D. before pneumonectomy and one year after induction of oleothorax. No overdistention was observed.

Case 5.—D. D., a 26-year-old woman, had a pneumonectomy for multiple excavation of the upper lobe of the left lung and a single large cavity just above the left diaphragm (Fig. 9). The patient did well on pneumothorax treatment of the space formerly occupied by the left lung. After four months a tuberculous empyema developed and the space was filled with mineral oil.

Unlike the previously discussed cases, this patient's single "good" lung before operation was smaller than predicted (Table V). The VC was 1,310 c.c. (predicted 1,570) and the residual volume was 370 c.c. (predicted 390 c.c.). The RV/TC × 100 ratio was normal, that is, the lung was not overdistended in the pulmonary midposition. The lung to be resected showed remarkably good function in spite of extensive cavitation. It was responsible for about one-fourth of the total ventilation and oxygen uptake at rest.

One week after operation the patient developed a spontaneous pneumothorax of the one remaining lung. At that time, the VC was only 590 c.c., and the total lung volume was only 800 cubic centimeters. Complete re-expansion was accomplished at the end of

TABLE V. PNEUMONECTOMY FOLLOWED BY OLEOTHORAX (PATIENT D. D.)

FUNCTION TEST	PREOPERATIVE					POSTOPERATIVE, PNEUMOTHORAX					OLEOTHORAX		
	BRON. SPIR. RIGHT	TOTAL	ONE LUNG PRED.	LEFT	RIGHT	1 WK.* RIGHT	2 WK. RIGHT	2 MO. RIGHT	3 MO. RIGHT	4 MO. RIGHT	6 MO. RIGHT	1 YR. RIGHT	1½ YR. RIGHT
MBC, L./min.	--	58	40	--	--	--	42	46	45	46	45	45	39
Upright VC, c.c.	--	1,980	1,570	--	--	--	1,250	1,510	1,490	1,500	1,500	1,500	1,450
Air velocity index	--	1.16	1.00	--	--	--	1.33	1.19	1.18	1.21	1.19	1.19	0.94
Walking ventil., L./min.	--	16	14	--	--	--	--	--	--	--	16	--	16
Walking index, %	--	27	34	--	--	--	38	35	36	35	36	37	36
Basal resp. rate	15	15	--	15	15	32	16	14	14	13	13	15	14
Tidal volume, c.c.	72%	430	--	110	320	210	410	370	330	370	330	415	320
Inspir. capacity, c.c.	77%	1,360	--	310	1,050	590	870	1,015	1,000	980	1,110	1,015	965
Expiratory reserve, c.c.	76%	340	--	80	260	0	140	210	210	200	225	230	225
Vital capacity, c.c.	77%	1,700	1,570	390	1,310	590	1,010	1,225	1,210	1,180	1,335	1,245	1,190
Residual volume, c.c.	69%	540	390	170	370	210	430	585	570	580	625	615	560
Total lung capacity, c.c.	75%	2,240	1,960	560	1,680	800	1,440	1,810	1,780	1,760	1,960	1,860	1,750
RV/TC x 100, %	22	24	20	30	22	26	30	32	32	33	32	33	32
Ventilation, L./min.	72%	6,440	--	1,800	4,640	6,510	6,570	5,170	4,580	4,780	4,310	6,220	4,490
Oxygen uptake, c.c./min.	78%	201	--	42	159	217	212	196	203	201	192	200	192
Ventil. equiv., L./100 c.c.	2.9	3.3	2.2	4.3	2.9	3.0	3.1	2.6	2.2	2.4	2.2	3.1	2.3
Mixing index, % N_2	2.90	2.68	--	2.90	1.70	--	1.20	--	1.10	1.42	1.67	1.95	1.30

*Spontaneous pneumothorax of remaining lung.

138 per cent of the "one lung" predicted, and the VC was 94 per cent of this same value.

Pneumothorax completely confined the remaining lung within its normal position and volume, and it did so without causing any further reduction of maximal ventilation.

3. *Oleothorax.*—Two patients were maintained on pneumothorax for four and nine months, respectively, and the space was then filled with oil. This was done in one patient because of the appearance of clear yellow fluid containing *M. tuberculosis*, and in the other, because of appearance of sterile cloudy fluid. A third patient, not included in this series, has been followed for three years with oleothorax on one side and an extrapleural pneumothorax of the remaining lung.[5] The oil in the first two cases was mineral oil, and in the last case, olive oil. Formation of fluid subsided in the first two patients shortly after introduction of the oil, and all three patients are well at present.

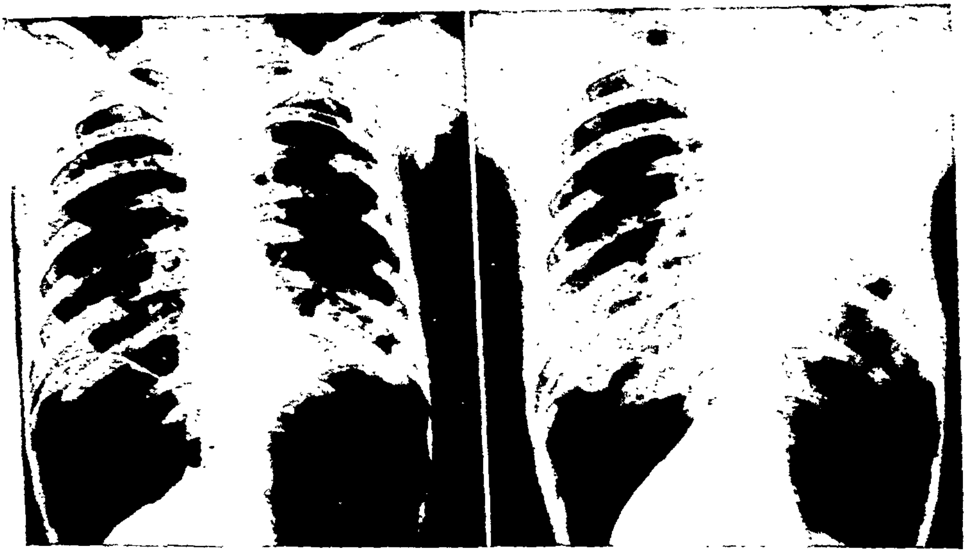

Fig. 9.—Roentgenograms of the chest of patient D. D. before pneumonectomy and one year after induction of oleothorax. No overdistention was observed.

Case 5.—D. D., a 26-year-old woman, had a pneumonectomy for multiple excavation of the upper lobe of the left lung and a single large cavity just above the left diaphragm (Fig. 9). The patient did well on pneumothorax treatment of the space formerly occupied by the left lung. After four months a tuberculous empyema developed and the space was filled with mineral oil.

Unlike the previously discussed cases, this patient's single "good" lung before operation was smaller than predicted (Table V). The VC was 1,310 c.c. (predicted 1,570) and the residual volume was 370 c.c. (predicted 390 c.c.). The RV/TC × 100 ratio was normal, that is, the lung was not overdistended in the pulmonary midposition. The lung to be resected showed remarkably good function in spite of extensive cavitation. It was responsible for about one-fourth of the total ventilation and oxygen uptake at rest.

One week after operation the patient developed a spontaneous pneumothorax of the one remaining lung. At that time, the VC was only 590 c.c., and the total lung volume was only 800 cubic centimeters. Complete re-expansion was accomplished at the end of two weeks. At that time, the usual immediate postoperative changes were observed. After two months, the VC had regained its preoperative value but the residual volume had increased to 585 c.c., and the RV/TC ratio was elevated to 30 per cent. Thus, at deepest inspiration, the lung was still smaller than the single predicted lung volume, but during quiet breathing the lung was slightly overdistended. No further change occurred during the next two months. At the end of four months, the space was filled with oil (Table V). *This caused no further change of maximal ventilation* as expressed by the MBC. The total lung capacity also remained entirely unchanged and there was no alteration in the relationship of the subdivisions of the total capacity. No further change occurred during the course of the next one and one-half years.

Oleothorax replacing pneumothorax as lung prosthesis caused no further change of maximal ventilation and no alteration of the total capacity and its subdivisions.

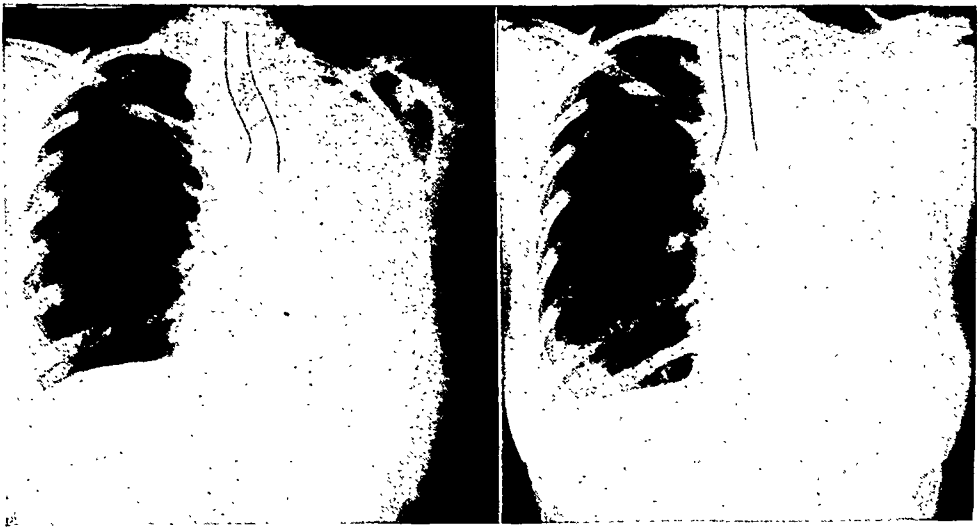

Fig. 10.—Roentgenograms of the chest of patient F. Z. before pneumonectomy and four months after introduction of a plastic sponge into the space. The overdistention existing before operation has been corrected.

4. *Plastic Sponge Plombage.*—In three patients, the space was maintained as a pneumothorax for three to nine months, and a plastic sponge prosthesis was then introduced. The sponge (Ivalon) was used by Grindley and Clagett in experimental animals and was found to be relatively inert.[23] Its use in man has not been recorded previously. From one to three sponges were shaped roughly to fit the space, soaked in a penicillin-streptomycin solution and introduced through the fourth intercostal space. A small amount of fluid was removed on a few occasions during the first two weeks after operation. The spaces then remained dry, and the structural detail of the sponges was visible on the roentgenogram.

Case 6.—F. Z., a 24-year-old woman, was admitted with a completely atelectatic left lung (Fig. 10). Severe ulcerative endobronchial disease was noted on bronchoscopy of the left mainstem bronchus, and the bronchus was completely occluded by scarring, stricture, and edema proximal to the take-off of the left upper lobe. The left lung was removed, and the space was maintained as a pneumothorax for two months. At that time the major portion of three sponges, measuring 3 by 4 by 7 inches, was introduced into the space. The postoperative course was uneventful.

Both the preoperative roentgenogram (Fig. 10) and the preoperative lung volume studies (Table VI), showed marked overdistention of the functioning lung. This overdistention, however, had not taken place in the residual volume fraction of the total capacity. All components of the total capacity were proportionately enlarged, and the RV/TC ratio was therefore normal. This patient, like A. L. (Table IV), had a large well-functioning lung.

The changes observed immediately after operation and during the two months of pneumothorax treatment did not differ from those of patient A. L. Just prior to plombage the total capacity and the MBC were slightly smaller than before operation, and the residual volume was slightly larger than before.

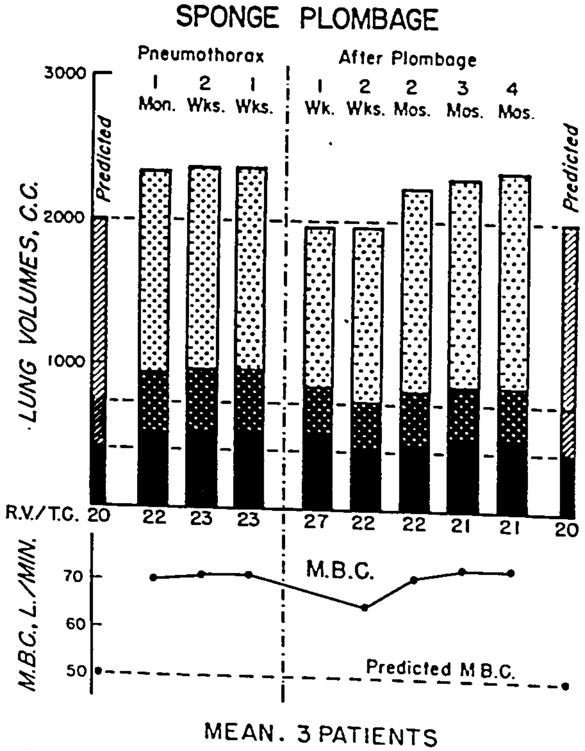

Fig. 11.—Mean function studies of three patients in whom pneumonectomy was followed by pneumothorax treatment of the space and later plastic sponge plombage.

Introduction of the sponges caused a decrease of MBC and all the components of the total capacity for only two weeks. Two months after secondary operation, the MBC, VC, and total capacity had regained the values for the single lung found before pneumonectomy. Only the residual volume remained decreased and the RV/TC × 100 percentage of 14 was the lowest recorded in this series. This was found to be due to compression of the lung by accumulation of fluid around the sponge. This fluid had never been removed after operation, and its removal now brought a return of the residual volume to its normal value seen at the end of three months.

The results of studies of three patients with sponge prostheses are summarized in Fig. 11. The changes caused by plombage are what the physiologist might consider as "ideal." The stroke volume as expressed by the vital capacity and the maximal ventilation as expressed by the MBC remained unchanged, and

TABLE VI. PNEUMONECTOMY FOLLOWED BY PNEUMOTHORAX AND LATER PLASTIC SPONGE PLOMBAGE (PATIENT F. Z.)

FUNCTION TEST	BEFORE OP.		PNEUMONECTOMY & PNEUMOTHORAX					SPONGE PLOMBAGE			
	ONE LUNG PRED.	RIGHT*	1 WK. RIGHT	1½ WK. RIGHT	2 WK. RIGHT	1 MO. RIGHT	2 MO. RIGHT	1 WK. RIGHT	2 WK. RIGHT	2 MO. RIGHT	3 MO. RIGHT
MBC, L./min.	58	60	48	50	58	59	55	--	52	58	60
Upright VC, c.c.	1,705	2,350	2,010	2,030	2,380	2,320	2,290	--	1,920	2,110	2,300
Air velocity index	1.00	0.67	0.70	0.72	0.71	0.73	0.70	--	0.81	0.80	0.84
Walking ventil. L./min.	20	28	--	--	--	--	--	--	--	--	--
Walking index, %	34	47	58	56	48	48	51	--	54	48	47
Basal resp. rate	--	14	17	18	14	13	18	26	22	21	21
Tidal volume, c.c.	--	480	440	430	445	460	355	320	290	345	350
Inspir. capacity, c.c.	--	1,710	1,450	1,450	1,640	1,830	1,600	1,330	1,390	1,820	1,920
Expiratory reserve, c.c.	--	550	405	410	420	350	350	270	225	290	350
Vital capacity, c.c.	1,705	2,260	1,855	1,860	2,060	2,180	1,950	1,600	1,615	2,110	2,270
Residual volume, c.c.	425	510	655	660	600	410	565	435	295	350	570
RV/TC × 100, %	20	18	26	26	23	15	22	21	15	14	20
Total capacity, c.c.	2,130	2,770	2,510	2,520	2,660	2,590	2,515	2,035	1,910	2,460	2,840
Ventilation, L./min.	--	6,800	7,520	7,680	6,300	6,150	6,375	8,300	6,360	7,250	7,230
Oxygen uptake, c.c./min.	--	342	238	347	236	233	255	297	230	234	232
Vent. equiv., L./100 c.c.	2.2	2.0	3.2	2.2	2.7	2.6	2.5	2.8	2.8	3.1	3.1
Mixing index, % N_2	--	1.24	2.70	1.97	1.74	1.30	1.67	0.69	0.68	0.65	0.77

*No patent left main-stem bronchus.

the residual volume was decreased sufficiently to make its ratio to the total capacity normal.

Plastic sponge plombage caused no reduction of maximal function, and confined the remaining lung within its normal space. Preoperative overdistention could be corrected by this procedure. The follow-up period was only brief.

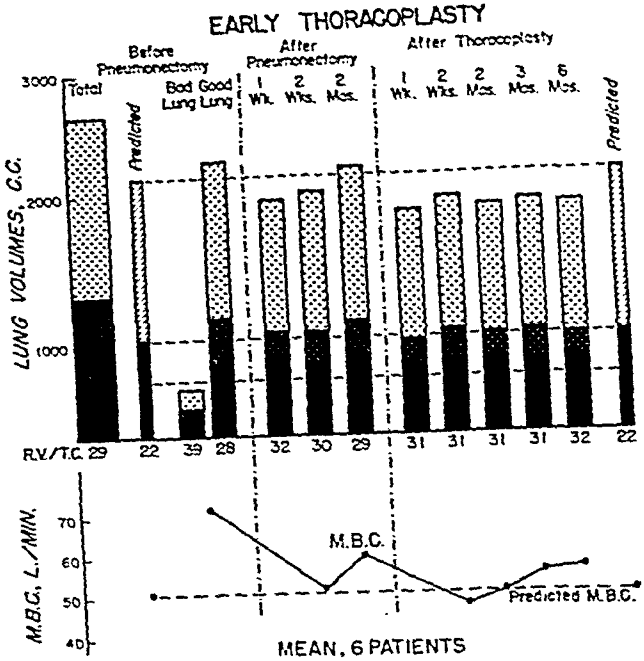

Fig. 12.—Mean function studies of six patients in whom pneumonectomy was followed by thoracoplasty six weeks to two months later.

5. *Early Thoracoplasty.*—This procedure was carried out in six patients six weeks to two months after resection. Portions of five to seven ribs were removed in one stage, not including the rib resected at the time of pneumonectomy. The first rib and the transverse processes were not disturbed to minimize deformity.

The laboratory findings in six patients before pneumonectomy, immediately after resection and after thoracoplasty are summarized in Fig. 12. The lung to be resected contributed only 20 per cent of the total lung volume before operation. The "good" lung before operation was slightly overdistended both in full inspiration and in the midposition. Changes in lung volumes and MBC during the first two months after operation differed in no way from the groups discussed previously and might be superimposed on Fig. 6, showing changes of pneumonectomy without aftertreatment. The MBC was reduced from 72 to 52 liters per minute immediately after resection, and rose to 60 liters per minute at the end of two months.

Early thoracoplasty further reduced the MBC to 48 liters per minute and six months after this operation, it had risen to 56 liters per minute, but never

TABLE VII. PNEUMONECTOMY FOLLOWED BY LATE THORACOPLASTY (PATIENT A. P.)

FUNCTION TEST	PREOPERATIVE		AFTER PLEURO-PNEUMONECTOMY				AFTER THORACOPLASTY				
	ONE LUNG PRED.	RIGHT*	1 WK. RIGHT	2 WK. RIGHT	2 MO. RIGHT	4 MO. RIGHT	1 WK. RIGHT	2 WK. RIGHT	2 MO. RIGHT	4 MO. RIGHT	1½ YR. RIGHT
MBC, L./min.	41	50	--	43	44	45	--	35	35	35	31
Upright VC, c.c.	1,430	2,030	--	1,530	1,700	1,770	--	1,530	1,540	1,540	1,210
Air velocity index	1.00	0.86	--	0.96	0.91	0.84	--	0.81	0.81	0.81	0.90
Walking ventilation, L./min.	15	17	--	--	--	--	--	--	--	--	--
Walking index, %	36	34	--	40	38	36	--	49	49	49	55
Basal resp. rate	--	21	26	22	21	24	30	28	24	23	23
Tidal volume, c.c.	--	245	260	260	240	260	190	180	210	230	220
Inspir. capacity, c.c.	--	1,520	1,050	1,250	1,330	1,290	1,175	1,150	1,165	1,180	1,130
Expiratory reserve, c.c.	--	230	160	200	220	255	200	220	210	220	195
Vital capacity, c.c.	1,430	1,750	1,210	1,450	1,550	1,545	1,375	1,370	1,375	1,400	1,325
Residual volume, c.c.	435	1,070	770	1,150	1,170	1,020	645	710	775	790	900
Total capacity, c.c.	1,865	2,820	1,980	2,600	2,720	2,555	2,020	2,080	2,150	2,190	2,225
RV/TC × 100, %	23	38	39	44	43	40	32	34	36	36	40
Ventilation, L./min.	--	5,120	6,700	5,700	5,040	6,250	5,700	5,040	5,040	5,290	5,020
Oxygen uptake, c.c./min.	--	210	249	228	242	250	275	260	235	220	230
Ventil. equiv., L./100 c.c.	2.2	2.4	2.7	2.5	2.1	2.5	2.1	1.9	2.1	2.4	2.2
Mixing index, % N_2	--	1.46	1.03	2.01	3.26	2.80	2.02	2.07	1.86	2.01	2.10

*No patent left main-stem bronchus.

regained the value found just prior to thoracoplasty. All components of the total lung capacity were permanently reduced by this secondary operation. The remaining lung six months after thoracoplasty, was smaller than the predicted. The overdistention at full inspiration was thus overcorrected. However, in the pulmonary midposition, the lung was still overdistended in spite of a reduction of the expiratory reserve. The RV/TC × 100 percentage of 32 was larger than immediately prior to thoracoplasty. Similar changes were observed in patient R. M. (Table II).

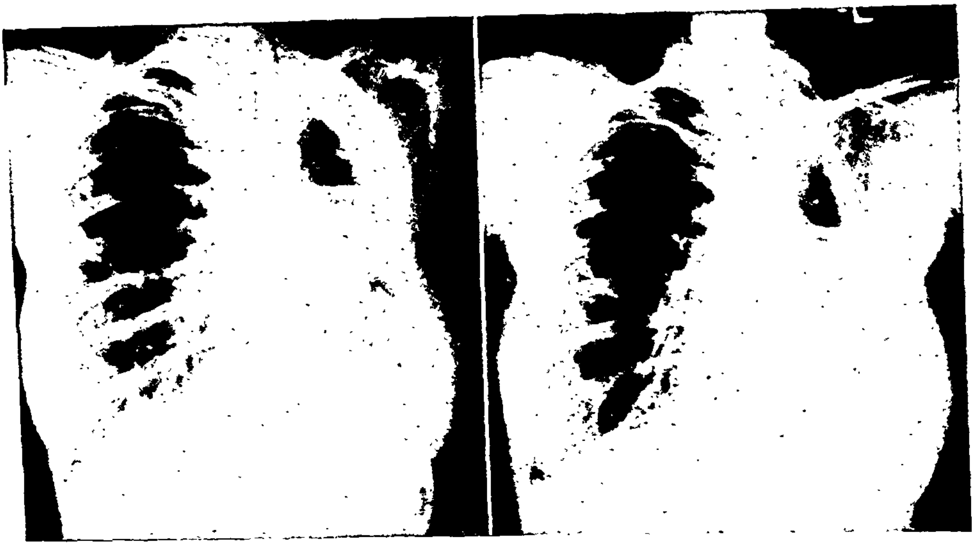

Fig. 13.—Roentgenograms of the chest of patient A. P. three months after pleuro-pneumonectomy and six months after thoracoplasty. The posterior herniation of the right lung was not corrected.

The alterations in function caused by early thoracoplasty were not entirely desirable. The maximal stroke volume as expressed by the VC was reduced, maximal ventilation tested by the MBC was permanently reduced and the residual volume still was larger than predicted. No overdistention was present on deepest inspiration, a respiratory position assumed relatively rarely. Overdistention was present in the pulmonary midposition, the size in which the lung is most frequently encountered during a sedentary life.

6. *Late Thoracoplasty.*—This operation was done in one stage five months to two years after pneumonectomy. The first rib and the transverse processes were not removed. Resection of five to nine ribs including revision of the regenerated rib removed at the time of pneumonectomy was carried out.

Case 7.—Patient A. Z., a 36-year-old woman was admitted to the Sanatorium Division with a history of tuberculous empyema following unsuccessful pneumothorax ten years ago. An acute illness characterized by expectoration of much foul sputum and high fever began one week before admission. A diagnosis of putrid empyema with bronchopleural fistula was made and the space was drained. Bronchoscopy revealed only a pin point opening of the left main-stem bronchus. Four months after admission, the entire parietal pleura, including the lung, was removed. A posterior herniation of the contralateral lung was noted at operation. The patient made an uneventful recovery except for a permanent Horner's syndrome first noted immediately after operation.

Physiologic data are shown in Table VII. Before operation, both VC and MBC were somewhat larger than predicted. The total capacity was greatly elevated due to a residual volume of the single lung which was larger than that predicted for two normal lungs. This residual volume occupied 38 per cent of the total capacity (RV/TC × 100). The various components of the total lung capacity were reduced only at the one week interval after operation. At the end of four months, the VC was smaller than before operation but the residual volume had regained its former value and was now 44 per cent of the total capacity.

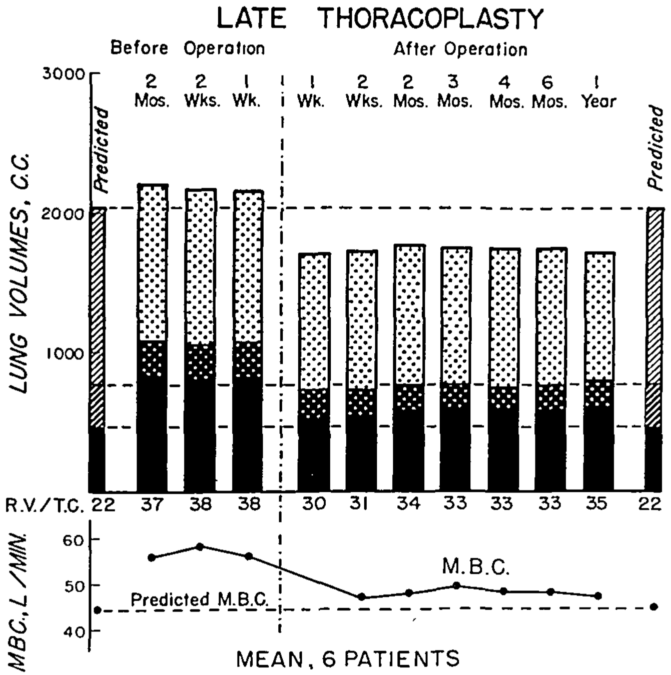

Fig. 14.—Mean function studies of six patients in whom pneumonectomy was followed by thoracoplasty six months to two years later.

Roentgenograms of the chest at this time revealed a large posterior herniation of the right lung which was also observed at the time of operation (Fig. 13). Because of the very high position of the diaphragm, a small thoracoplasty was performed. Both the physiologic data (Table VII) and the roentgenogram (Fig. 13) showed no or very slight reduction of this herniation. The lung remained overdistended both on full inspiration and in the pulmonary midposition. One and one-half years later, the residual volume still occupied 40 per cent of the total capacity.

The changes of VC and MBC after late thoracoplasty were in great contrast to those after oil and sponge plombage. There was a slight permanent reduction of stroke volume but maximal voluntary ventilatory ability, as measured by the MBC, was severely and permanently reduced from 45 to 35 liters per minute. A loss of ten liters of MBC is of no significance if the initial MBC is 100 or even 200 liters per minute. Here ten liters represented a 22 per cent decrease and the patient who had never been dyspneic before thoracoplasty, now complained of exertional dyspnea. The further reduction to 31 liters one year later may well represent the onset of true emphysema with early disruption of the pulmonary parenchymal architecture.

Fig. 14 shows the rather striking mean findings of six patients after late thoracoplasty. Two months, two weeks, and one week before thoracoplasty, the lung was moderately overdistended both on full inspiration and in the pulmonary midposition, more so in the latter. The residual volume was nearly twice normal and occupied 38 per cent of the total capacity. Thoracoplasty accomplished just the opposite of the desired result. The range of the useful respiratory movement (VC) was greatly reduced, whereas the volume which merely dilutes the inspired tidal air (residual volume) was only slightly reduced. It still occupied one-third of the total capacity. Worst of all, the initial mean MBC of 58 liters per minute was permanenty reduced to 46 liters per minute.

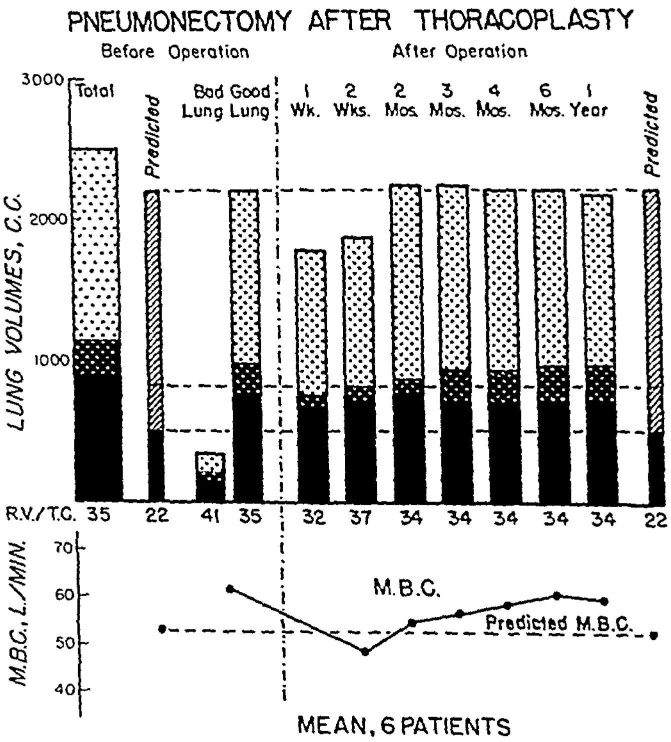

Fig. 15.—Mean function studies of six patients who had pneumonectomy after thoracoplasty failed.

Late thoracoplasty corrected overdistention at maximal inspiration chiefly at the expense of the vital capacity. It corrected overdistention on fullest expiration only partially. It caused a definite and permanent loss of maximum breathing capacity. These are striking findings since the thoracoplasty was done over an empty hemithorax. Possible causes for this will be discussed subsequently.

7. *Pneumonectomy After Thoracoplasty.*—The lung was removed in six patients after thoracoplasty had failed. Thoracoplasty had been performed from two to thirteen years prior to resection, and had been extensive (eight to ten ribs). Thoracoplasty failures with fewer ribs removed usually had secondary lobectomies.

Findings after this procedure, summarized in Fig. 15, can be stated briefly. Except for the immediate postoperative period, pneumonectomy after extensive thoracoplasty had no effect on the total capacity or MBC of the single remaining lung. There was no overdistention in full inspiration either before or after resection and there was a moderate overdistention in the pulmonary midposition both before and after. The functional residual capacity was increased and the residual volume occupied 35 per cent of the total capacity before operation and 34 per cent two months to one year after resection.

Thoracoplasty Versus Pneumonectomy for the Functionless Lung.—In the course of a detailed study of the ventilatory defect due to thoracoplasty, it was noted that this procedure caused a considerable permanent loss of function even if performed over an apparently functionless lung.[24] Ten patients were selected from this group whose bronchospirometry prior to operation showed less than 10 per cent of the total resting ventilation on the side to be operated. Seven of these patients had total atelectasis due to empyema or therapeutic pneumothorax. Three patients had cavernous disease involving the entire lung. Their individual MBC and upright VC before operation, two weeks after every one of three stages of thoracoplasty, and six months after thoracoplasty are shown in Fig. 15. Their mean MBC before operation was 71 liters per minute and six months later 56 liters, a permanent loss of 21 per cent of maximal ventilatory capacity. The mean VC was 2,350 c.c. before operation and 1,725 c.c. six months later, a 27 per cent loss of maximal stroke volume.

In the present series of patients, pneumonectomy was performed for "destroyed lung" in twelve instances. By bronchospirometry, nine patients showed less than 10 per cent of the total ventilation on the side to be operated. Two others showed no patency of the mainstem bronchus by bronchoscopy and no aerated lung on that side by roentgenogram. The last patient had ulcerative endobronchial disease with stricture of the mainstem bronchus. The MBC and upright VC of this group before operation, two weeks after operation, and every month thereafter for six months are also shown in Fig. 16. Their mean MBC was 58 liters per minute before operation and 57 liters per minute six months later, a loss of only 2 per cent. The mean VC at the same time intervals was 1,675 c.c. and 1,625 c.c., again a decrease of only 3 per cent.

Our clinical experience supported these data. There were several patients in the thoracoplasty group who were not dyspneic with one functioning lung before collapse and who had moderate dyspnea six months after thoracoplasty over a totally atelectatic lung. A number of these patients had total empyema of many years' duration with marked emphysema of the remaining lung. They often had considerable respiratory difficulty, and one such patient died of respiratory failure shortly after a first-stage thoracoplasty.

Several patients who had a nonfunctioning lung removed were moderately dyspneic before operation. One woman, before pneumonectomy, had an MBC of 32 liters per minute or 29 per cent of predicted and a VC of 820 cubic centimeters. Although she required oxygen inhalation therapy for about two weeks after resection, she was less dyspneic six months after the operation than before resection. Her MBC at that time was 34 liters per minute and her VC 880 cubic centimeters. She has worked as a full-time secretary for the past two years.

The laboratory data and clinical experiences were further underlined by the lung volume and maximal function studies before and after thoracoplasty of a hemithorax from which the lung had been previously removed (Tables II and VII, Figs. 12 and 14). Almost invariably, this secondary procedure caused a further and permanent loss of maximal function. This was most strikingly demonstrated in the group who had thoracoplasty late after pneumonectomy, because the patients had had opportunity fully to recover from the resection (Fig. 14).

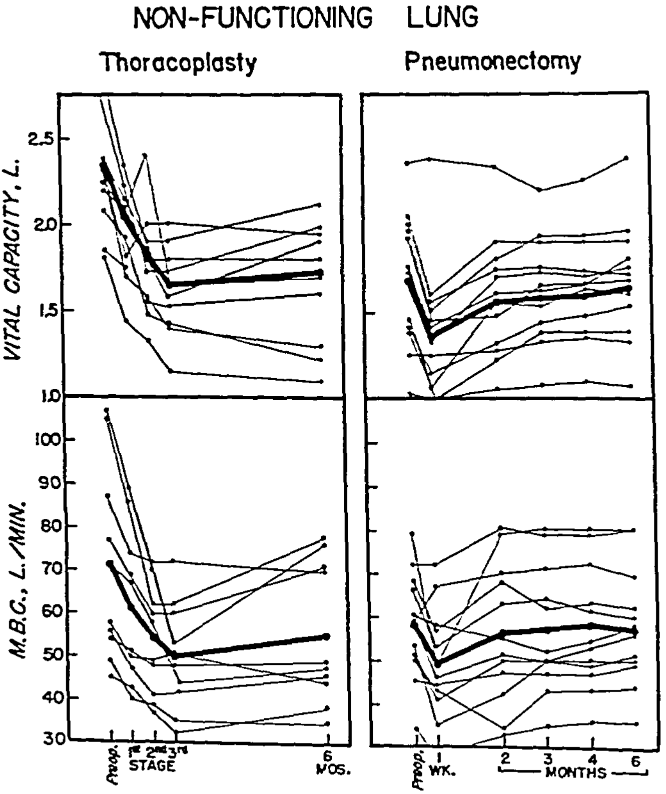

Fig. 16.—Serial maximum breathing capacity and vital capacity studies in patients who had primary thoracoplasty or primary pneumonectomy of lungs which showed less than ten per cent of the total function prior to operation.

It must be concluded that thoracoplasty over a functionless lung usually causes a permanent loss of function whereas resection of such a lung usually does not. It might be argued that the functional effectiveness of the diseased lung prior to collapse was underestimated for one reason or another. However, the studies of the effect of thoracoplasty of an empty hemithorax prove that destruction of an extensive portion of the hemithorax impairs the functional efficiency of the contralateral lung.

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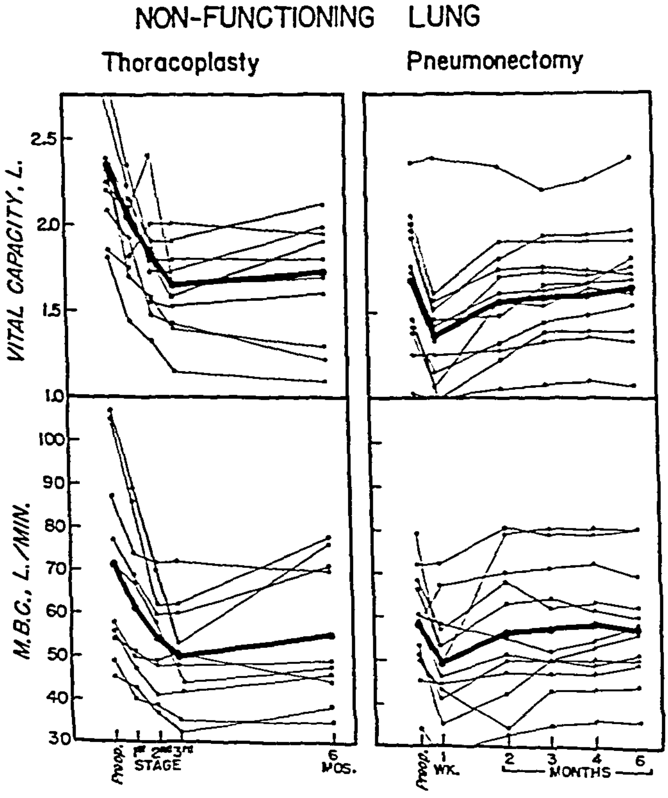
NON-FUNCTIONING LUNG

Thoracoplasty

Pneumonectomy

Fig. 16.—Serial maximum breathing capacity and vital capacity studies in patients who had primary thoracoplasty or primary pneumonectomy of lungs which showed less than ten per cent of the total function prior to operation.

It must be concluded that thoracoplasty over a functionless lung usually causes a permanent loss of function whereas resection of such a lung usually does not. It might be argued that the functional effectiveness of the diseased lung prior to collapse was underestimated for one reason or another. However, the studies of the effect of thoracoplasty of an empty hemithorax prove that destruction of an extensive portion of the hemithorax impairs the functional efficiency of the contralateral lung.

Two mechanisms, both originating with the postural changes due to thoracoplasty may be responsible for the loss of function. The proper functioning of the muscles of respiration depend in part on the application of symmetrical leverage. This is perhaps illustrated by the occasional spontaneous fracture of the first contralateral rib after a thoracoplasty in the course of which, the accessory muscles of respiration in the neck have been severed from their insertions to the ribs. When a contraction of some respiratory muscles is no longer counterbalanced by an equal contralateral contraction, their action becomes less efficient.

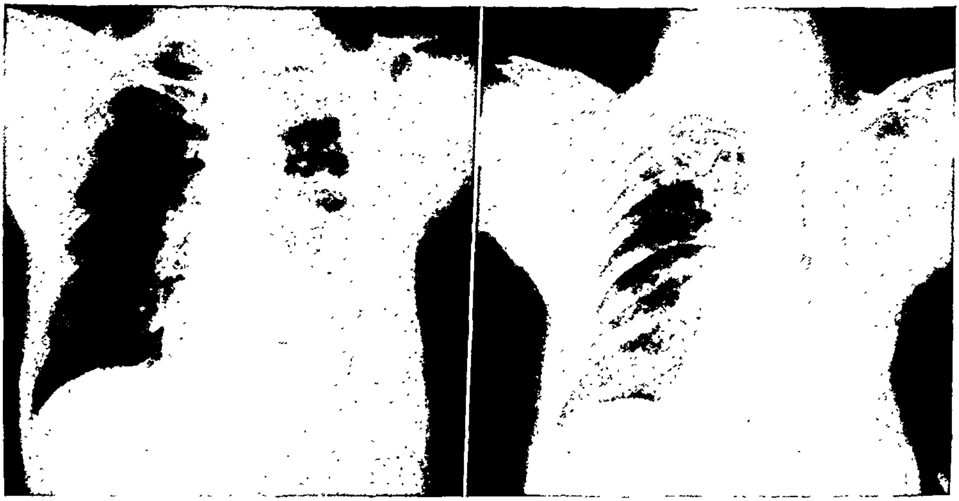

Fig. 17.—Roentgenograms of the chest of patient J. O'K. before and one year after a standard ten-rib thoracoplasty. The alteration of the size and shape of the contralateral hemithorax is illustrated.

The scoliosis is, in part, due to the unequal pull of the respiratory muscles, and, in part, to relaxation and interruption of the costospinal muscle groups. This scoliosis may be responsible for the second mechanism of functional reduction. The thoracic cage on the contralateral side becomes partly "jackknifed." This not only reduces the functional residual volume of the remaining lung, but all other components of the lung volume as well (Fig. 14).

The maximal effort is impaired by less effectively functioning respiratory muscles and the stroke volume is reduced by "jack-knifing" of the rib cage. This is illustrated by the roentgenograms of Fig. 16. This patient was admitted with a left pyopneumothorax. Bronchospirometry showed no oxygen uptake and no respiratory excursions on that side. A two-stage, ten-rib thoracoplasty caused moderate scoliosis, a well-marked "jackknifing" of the contralateral thoracic cage (Fig. 17) and a loss of MBC of 30 per cent, which was not regained during the first year after operation.

In patients with marginal pulmonary function, thoracoplasty should be approached with caution even if there is no lung or a destroyed lung on the side to be collapsed.

CONCLUSIONS

There is no preponderance of evidence that chronic overdistention leads to true pulmonary emphysema with the histologic changes characterized by disruption of the pulmonary parenchymal architecture. Only one of the physiologic manifestations of emphysema, an increased ratio of residual volume to total lung capacity (RV/TC) was present in many patients who showed marked overdistention. Additional characteristics of emphysema, such as reduced timed vital capacity percentages, elevated intrapulmonary mixing indices, low immobile diaphragms, decreased arterial oxygen saturation, and increased arterial and alveolar carbon dioxide tension were all absent in patients who had not shown these changes prior to resection. The total predicted and determined vital capacities, together with the volumes expired during the first, first two, and first three seconds of the effort are shown for thirty-five normal volunteers and patients with pneumonectomy without aftertreatment (Fig. 18). Patients with one lung showed a severe restrictive ventilatory insufficiency. The stroke volume (VC) was reduced to almost one-half of the predicted for two lungs. However, there appeared to be a slight obstructive element also. Whereas normal volunteers were able to exhale 83, 94, and 97 per cent of their total vital capacity in the first, first two, and first three seconds of the effort, patients after pneumonectomy exhaled only 67, 77, and 79 per cent of their total vital capacity at the respective time intervals.[18] In only a few patients was there a slight progressive decrease of the MBC, two and two and one-half years after operation. In one patient, there was good evidence of rapid progression of pre-existing em-

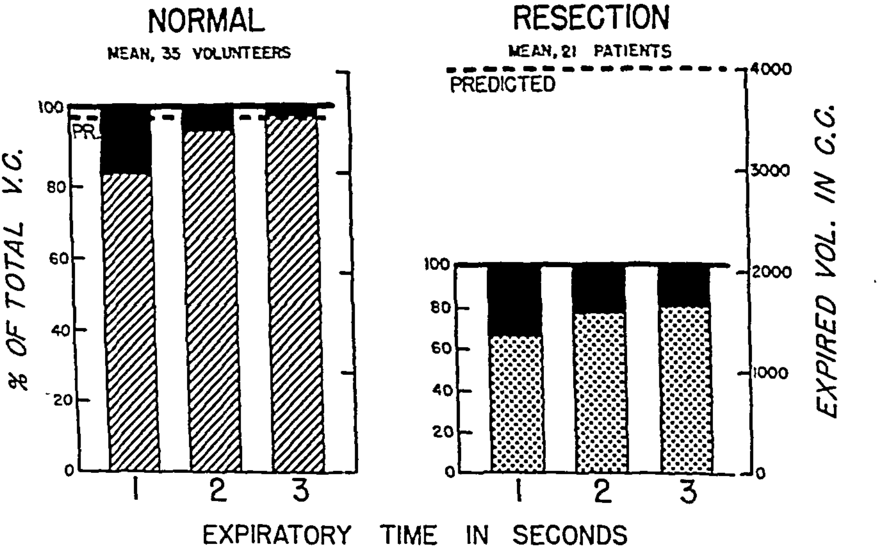

Fig. 18.—Predicted, total determined and timed vital capacities in normal volunteers and in patients with pneumonectomy without aftertreatment. The one-second, two-second, and three-second capacities were obtained with a modified vital capacity instrument and an electronic timer.

physema. It is thought probable but not certain that pulmonary parenchymal disruption will eventualy be the end result of chronic overdistention.

Overdistention did not lead to true emphysema in young people after pneumonectomy within the first two and one-half years. In this respect, the study must be regarded as a preliminary report.

From the present investigation, it can be concluded that some overdistention of the remaining lung cannot be avoided if the empty hemithorax is not treated. It was shown that the extent of this overdistention is unpredictable and varies greatly from patient to patient. A prosthesis of oil, plastic sponge or air under slightly positive pressure obviated entirely overdistention of the remaining lung and mediastinal shift. Such prostheses caused no further loss of maximal ventilatory function. Thoracoplasty within three months after resection also prevented overdistention but did cause a definite additional loss of maximal ventilation. Thoracoplasty performed more than six months after resection caused only a partial return of the opposite lung to its normal confines, and also produced a very definite and permanent further loss of pulmonary function.

If it is accepted that overdistention is undesirable, then with prevention of overdistention as the only objective in mind, a clear choice of treatment following pneumonectomy can be made from information obtained in this study. Pneumothorax, later followed by plastic sponge plombage, offers the ideal answer to this objective.

However, prevention of overdistention should always be only one of many objectives all of which together can be summarized as the optimal well-being of the patient. The maintenance of a large air-filled dead space often exposed to tuberculous mediastinal lymph nodes and a bronchial stump with peribronchial and endobronchial disease should not be accepted as the procedure of choice without far greater experience and evaluation from the surgical viewpoint. Indeed, the authors strongly suspect that the incidence of empyema with this form of treatment is higher than with thoracoplasty or with no treatment.

Furthermore, the introduction of a plastic sponge, even though a relatively inert foreign body, is a surgically unattractive idea, particularly in young patients and in patients with tuberculosis.

At this time, we do not wish to be interpreted as advocating the use of these methods in patients in whom resection has been performed for tuberculosis except in very special circumstances. The observations made, however, can be transferred to the group of patients in whom resection is done for malignant tumors of the lung. In this group, pre-existing emphysema, marginal pulmonary function, a poor general condition, and a questionable prognosis all make supplementary thoracoplasty undesirable. These very conditions make prevention of overdistention highly important. Pneumothorax followed at a variable interval by introduction of a plastic sponge prosthesis or even primary plastic sponge plombage at the time of pneumonectomy may provide the ideal solution to this problem.

It is hoped that these data may serve as a base line for further studies directed at the conservation of pulmonary function and its maintenance at an optimal level following pneumonectomy.

SUMMARY

Forty patients were studied before pneumonectomy and at weekly and later monthly intervals thereafter, for six months to two and one-half years.

The techniques employed included standard pulmonary function studies, preoperative bronchospirometry, a modified method of residual volume determination permitting measurement of small changes in that value and a new method for determination of functional residual capacity and pulmonary mixing index for each lung separately before operation.

Pneumonectomy without aftertreatment was followed by a variable amount of overdistention. The severity of this overdistention could not be predicted before operation.

Maintenance of pneumothorax within the space after operation entirely prevented overdistention. This procedure was not accompanied by further reduction of maximal function.

Conversion of pneumothorax to oleothorax caused no alteration of pulmonary volumina or function.

Conversion of pneumothorax by a plastic sponge plombage prevented overdistention entirely and overdistention existing prior to pneumonectomy could be corrected by this procedure. Plombage was not accompanied by a further reduction of maximal ventilatory function.

Early thoracoplasty, six weeks to two months after resection, prevented overdistention on deep inspiration by reduction of the vital capacity but did not entirely prevent overdistention in the pulmonary midposition. This procedure caused a further loss of maximum breathing capacity.

Late thoracoplasty, six months to two years after resection, corrected existing overdistention only partially and caused a further, permanent reduction of vital capacity and maximum breathing capacity.

Pneumonectomy after unsuccessful, extensive thoracoplasty produced no change of pulmonary volumina or maximal ventilatory function.

Pulmonary function before and after primary pneumonectomy and primary thoracoplasty for functionless lungs were compared. It was shown that primary resection in these patients caused no further loss of function while the loss of function after thoracoplasty over functionless lungs was marked.

No definite indications were obtained that overdistention following pneumonectomy in young patients results in true emphysema with disruption of the pulmonary parenchymal architecture during the first two and one-half years after operation. Progression of true emphysema after pneumonectomy in the older patients was demonstrated.

The authors are indebted to Miss D. M. Donnelly and Miss S. S. Smith for their technical assistance.

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PULMONARY FUNCTION IN TRAUMATIC HEMOTHORAX TREATED BY DECORTICATION

Colonel James H. Forsee, Medical Corps, United States Army, Major Stephen L. Kylar, Medical Corps, Army of the United States (By invitation), and Major Hu A. Blake, Medical Corps, United States Army (By invitation),
Denver, Colo.

MANY members of this organization have participated in the valuable contributions relating to pulmonary decortication in the treatment of traumatic hemothorax.[1-13] The principles and practices evolved during the experience of World War II are the fundamentals of its present-day management. Recently, the Korean conflict has resulted in the concentration of a relatively large number of casualties suffering from thoracic injuries at Fitzsimons Army Hospital. Traumatic hemothorax was a concern of major significance, early treatment had been excellent, and one phase not previously studied was, within certain limitations, observed among these casualties. I refer specifically to pulmonary function studies in patients treated by decortication. The number of patients available in the early months of the campaign exceeded our facilities for functional studies, and many did not have pre- and postoperative functional evaluations. Exhaustive studies as described by Cournand and his associates, Wright, and others were not at the time feasible.[14-17] Data presented pertain only to certain preoperative and early postoperative functional findings. Factual observations will be detailed, certain interpretations will be suggested, but conclusions which these data may warrant will probably best be made by those with greater experience in this field of pulmonary dynamics.

MATERIAL

From among thirty-two casualties treated by pulmonary decortication for traumatic hemothorax during the period Sept. 26, 1950, to March 1, 1951, nineteen have had certain pulmonary function studies made including external spirometry and bronchospirometric determinations. This presentation is limited to the data obtained relative to the maximum breathing capacity noted by external spirometry and divided functional studies emphasizing oxygen consumption, vital capacity, and ventilation, as observed in these nineteen patients. All were young soldiers in physically good condition prior to their injury. Their ages varied from 18 to 31 years, and averaged 21 years. All were operated upon relatively early following injury, the time interval varying from thirty to ninety-seven days, and averaging sixty days. Two of the group had had prior open drainage for a frank empyema. In five (18 per cent) the presence of intrapulmonary foreign bodies were in themselves considered

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as warranting thoracotomy for their removal. Streptokinase and streptodornase were employed in several instances with favorable response in liquefying the intrapleural material. Bronchoscopy was normal in all instances, and no evidence of prior chronic pulmonary disease was observed. The functional studies were made from twenty-one to one hundred forty days postoperatively; the average for the group being sixty-one days. All were wounded in Korea, and all but one have returned to military duty.

CLINICAL CONSIDERATIONS

Probably the majority of patients suffering from the effects of traumatic hemothorax induced by the missiles of war present few problems in determining indications for decortication. In others the indications may be less definite. Our observations and military expediency influenced us to approach this problem with an appreciation that decisive action as to whether or not decortication should be recommended was necessary. Along with others we have repeatedly noted that decortication may be effectively accomplished months or even years after the initial injury. It was believed that the best functional results would be obtained if decortication was presently accomplished in this group of casualties, rather than to defer the decision for later evaluation. It is probable that certain of the patients in this series would have continued to carry on normal activities without any appreciable interference of pulmonary function had surgery not been performed. In several instances the functional disturbances were not marked, the adaptive functions of the body had made a favorable adjustment as far as clinical observations could ascertain, and whatever small or moderate improvement which might result from surgical intervention would in such instances not be impressive. However, we know of no reliable means by which the development of moderate or severe chronic pulmonary insufficiency may be forecast in these individuals.

Pulmonary function studies were undertaken as a probable aid in assessing factors influencing the indications for or against surgical intervention. These data were often unpredictable. Careful fluoroscopic evaluation of the motion of the lung and diaphragm was carried out in all patients pre- and postoperatively.

CASE 1.—A Filipino, aged 20 years, was wounded Nov. 1, 1950, in the left thigh. He was behind enemy lines and remained hidden in the hills until captured ten days later. After being held captive overnight, he was instructed to walk toward a ditch. Upon reaching the ditch, he was shot by enemy rifle fire in the left chest. The patient remained in the ditch until nightfall, apparently being left as dead by the enemy. After dark he emerged from the ditch and hid in uninhabited houses until he was discovered two days later by American forces. Thoracentesis of the left pleural space was carried out on four different occasions with unknown amounts of bloody fluid being removed. He was admitted to Fitzsimons Hospital on Dec. 18, 1950. Approximately four-fifths of the left lung field was opaque on roentgenographic examination. The left diaphragm was immobile, there was no foreign body present, and on December 27 pulmonary decortication removing a visceral and parietal peel varying from 0.5 to 1 cm. in thickness was accomplished. His maximum breathing capacity preoperatively was 20 liters. Eighty-four days postoperatively it was recorded as 64 liters. Bronchospirometric determinations relative to the left lung showed oxygen consumption of 20 per cent prior to operation and 29 per cent postoperatively. Vital capacity increased from

13 per cent to 27 per cent, and ventilation showed 20 per cent increase. His diaphragm moved well following operation (Figs. 1 to 3).

CASE 2.—This patient, aged 20 years, was wounded in the left chest on Aug. 20, 1950. Hemoptysis was moderate. Three thoracentesis with removal of 800 c.c. of bloody fluid was accomplished prior to admission to Fitzsimons Hospital on September 26. Repeated

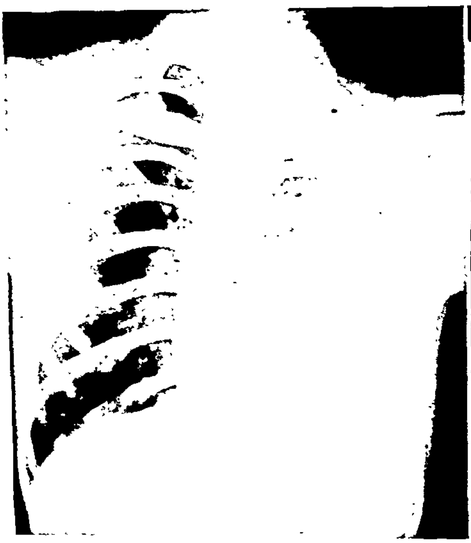

Fig. 1.

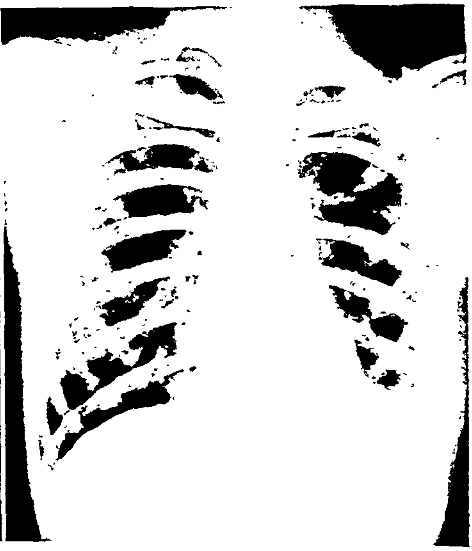

Fig. 2.

Fig. 1.—(Case 1) Traumatic hemothorax, left, seven weeks after initial injury.
Fig. 2.—(Case 1) Traumatic hemothorax, left, three weeks following decortication.

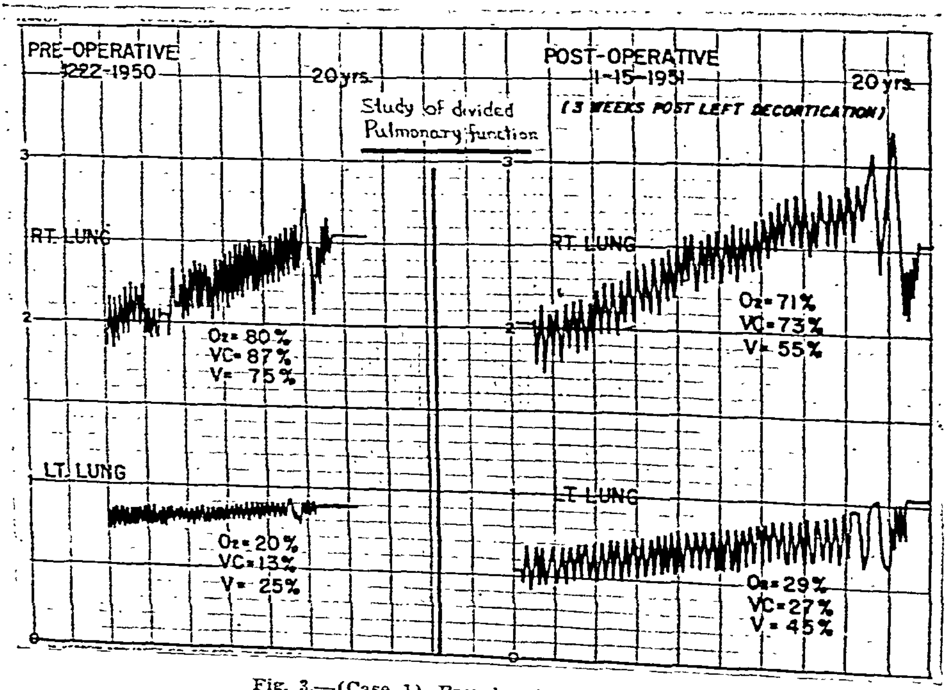

Fig. 3.—(Case 1) Bronchospirometric findings.

thoracentesis failed to accomplish re-expansion of the lung, and on October 23 pulmonary decortication was performed with the removal of two foreign bodies from the left lung. Maximum breathing capacity, preoperatively, was 68 liters, and seventy-seven days postoperatively it was recorded as 114 liters. Bronchospirometric determination relative to the left lung showed a gain of 9 per cent in oxygen consumption, a 7 per cent decrease in vital capacity and 3 per cent increase in ventilation on the seventy-seventh day postoperatively (Figs. 4 and 5).

Case 3.—Aged 18, wounded September 2 in the right chest, the missile traversing the right lung, lodging in the left lung. Repeated thoracentesis with removal of unknown amounts of bloody fluid on the right was accomplished prior to admission to Fitzsimons Hospital September 24. The bilateral nature of injury with a known hemothorax on the right and two foreign bodies on the left presented a problem of evaluation as to surgical intervention. The right diaphragm was immobile and the left diaphragm moved sluggishly. Maximum breathing capacity preoperatively was 37 liters. Decortication of the left lung and removal of foreign bodies were accomplished forty-seven days after injury, and twenty-eight days postoperatively functional studies revealed the following: maximum breathing capacity 72 liters, an increase of 35 liters over the preoperative determination. Oxygen consumption on the left was within normal limits preoperatively and decreased from 50 per cent to 47 per cent after operation. Vital capacity was 37 per cent prior to operation and increased 10 per cent after operation. Ventilation was 50 per cent preoperatively and decreased 6 per cent postoperatively.

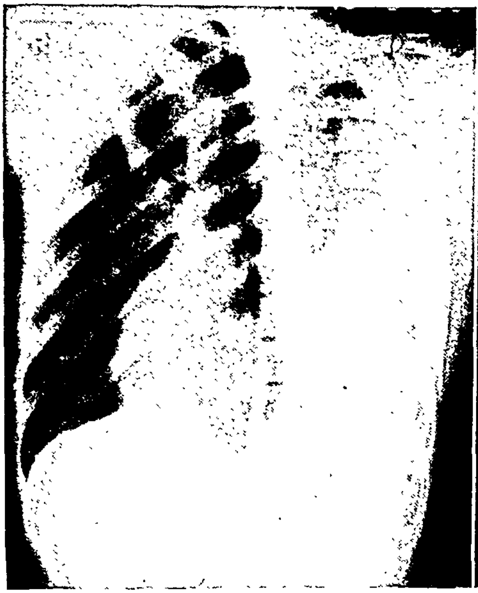

Fig. 4.

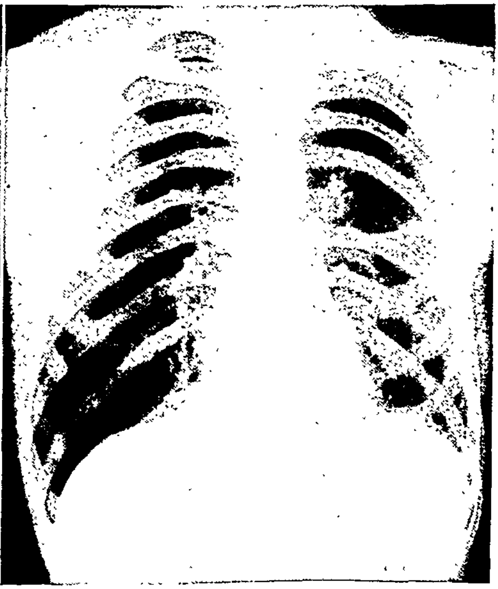

Fig. 5.

Fig. 4.—(Case 2) Traumatic hemothorax, left, ten weeks after initial injury. Intrapulmonary foreign bodies, left.

Fig. 5.—(Case 2) Twelve weeks after decortication and removal of foreign bodies.

Physiopathologic considerations resulting from a traumatic hemothorax due to the missiles of war have been thoroughly discussed elsewhere.[5, 6, 9] Simply stated as a working concept, it is our understanding that the pulmonary volume is reduced, and the encased or imprisoned lung is limited in participation in the act of breathing. The lung takes up less oxygen than it should, the pulmonary artery system responds by diminishing the blood flow through the lung attempting to prevent hypoxia, and a nonexpansile "peel" or membrane is formed as a result of the organization of blood and fluid in the pleural space. The degree to which these factors operate is variable, but is generally conceded to be progressive. The removal of the plombage of organized blood and exudate and the nonexpansile membrane should, it is believed, offer the best opportunity for re-expansion of the lung and return to as nearly as possible normal function. We have not been impressed by the problems or the results obtained as whether or not the parietal peel is removed. The parietal portion of the peel is generally removed but in several instances this peel has appeared to be of no particular significance and no special effort has been made to accomplish its removal. The time required for the maximum return of the function of the previously encased lung was not ascertained in our study. It would be an interesting observation to be able to carry out further functional studies on these or similar patients at intervals of several months or years. It is hoped that this may, in certain instances, be accomplished.

MAXIMUM BREATHING CAPACITY

Maximum breathing capacity is considered to be a most effective measurement of the bellow action of the chest, a reasonably accurate measurement of the patient's potential response to exertional stress, and a preferable method of evaluating respiratory reserve. Maximum breathing capacity measures the maximum amount of air moved in and out of the lungs per unit of time. In the sixteen patients in whom this observation was made by external spirometry, ten showed an increase postoperatively averaging 21 liters, while five showed a decrease averaging 15 liters and in one patient there was no change. The most striking changes were in Cases 1 and 2. The data on all patients are listed in Table I.

TABLE I. MAXIMUM BREATHING CAPACITY

SIDE INJURED	PREOPERATIVE	POSTOPERATIVE	PERCENTAGE INCREASE CHANGE	PERCENTAGE DECREASE CHANGE
L	70	105.6	36	
R	75	80	5	
L	70	70	0	
R	79.2	70.4		9
R	80.8	83.6	3	
L	88.4	67.6		21
L (operated) (bilateral injury)	37	72	35	
L	117.6	92		26
L	20	64	44	
L	91.4	96	5	
L	87.4	80.6		7
R	83.6	72		12
L	68.2	114.4	46	
R	76	96.8	21	
R	66	76	10	
L	67.2	71.6	4	

BRONCHOSPIROMETRY

Bronchospirometry permits divided functional studies of each lung being made at the same time. The percentages of oxygen consumption, vital capacity, and ventilation were made for each lung graphically by the closed method. Gas samples were not analyzed for oxygen and carbon dioxide. The variations pre-and postoperatively were appreciable. These data are illustrated in Tables II, III, and IV. The most marked increases in these factors were noted in two patients in whom the maximum breathing capacity decreased 9 liters in one, and increased 4 liters in the other, as illustrated in the following case report.

TABLE II. BRONCHOSPIROMETRIC DETERMINATION
(VENTILATION PERCENTAGES)

SIDE INJURED	PREOPERATIVE	POSTOPERATIVE	PERCENTAGE INCREASE CHANGE	PERCENTAGE DECREASE CHANGE
L	48.4	43		5
L	43	62	19	
R	44	50	6	
R	49.4	52	3	
L	50	52.5	3	
L	50	43.6		6
L	40	41.7	2	
L	37	54.5	18	
L	25	45	20	
R	46.5	56	10	
L	36.3	48	12	
L	44	39	5	
R	48.9	52.3	3	
L	39.6	42.9	3	
L	22	25	3	
R	40	48	8	
R	58	76	18	
L	29	40	11	

TABLE III. BRONCHOSPIROMETRIC DETERMINATION
(OXYGEN CONSUMPTION PERCENTAGES)

SIDE INJURED	PREOPERATIVE	POSTOPERATIVE	PERCENTAGE INCREASE CHANGE	PERCENTAGE DECREASE CHANGE
L	37.5	58.4	21	
R	68.8	50		19
L	47	40		7
R	50	72.3	22	
R	41	51	10	
L	61	46.8		14
L (operated) (bilateral injury)	50	47		3
L	34	50	16	
L	30	32	2	
L	20	29	9	
R	37.5	53	16	
L	24	46	22	
L	23	44	21	
R	38	60	22	
L	23	32.6	10	
L	25	45	20	
R	40	54.6	15	
R	42.8	40		3
L	20	45.4	25	

CASE 4.—This patient, aged 18 years, was wounded in the right chest by a hand grenade Sept. 13, 1950, and was admitted to Fitzsimons Hospital on October 1. A metallic foreign body was present in the right lower lung field and the right diaphragm was moderately elevated and immobile. Decortication with removal of the foreign body was accomplished Oct. 15, 1950. The "peel" was most adherent and thickest, as is the usual situation, over the diaphragm laterally and posteriorly. The maximum breathing capacity preoperatively was 79.2

liters, and eighty-four days postoperatively was 70.4 liters. The oxygen consumption on the right preoperatively was recorded as 50 per cent and increased to 72 per cent postoperatively. Vital capacity showed a 12 per cent increase, and the ventilation was increased 6 per cent (Figs. 6 and 7).

Case 5.—The patient, aged 24 years, was wounded in the left chest on Sept. 12, 1950. Several thoracenteses were performed with removal of an undetermined amount of blood prior to admission to Fitzsimons Hospital on October 2. Two foreign bodies were present in the

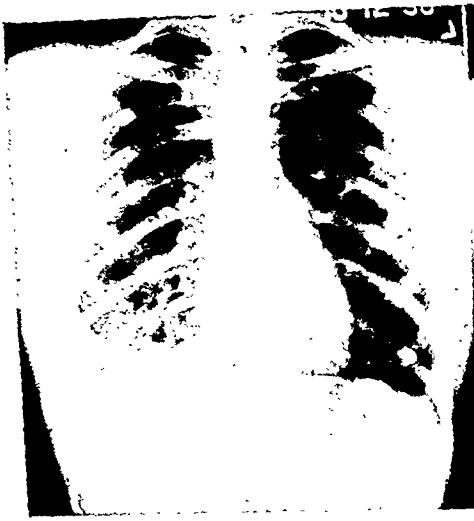

Fig. 6.

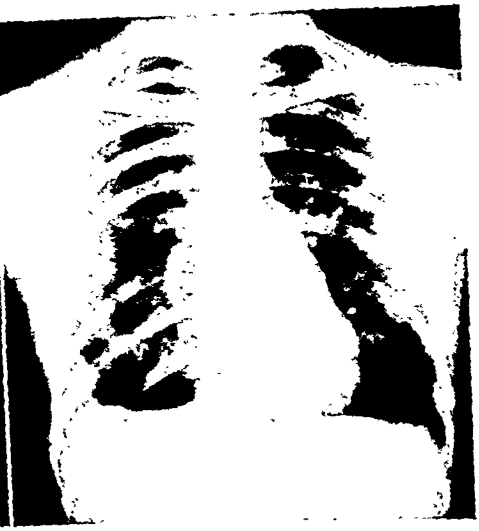

Fig. 7.

Fig. 6.—(Case 4) Traumatic hemothorax, right, four weeks after initial injury.
Fig. 7.—(Case 4) Traumatic hemothorax, right, twelve weeks after decortication.

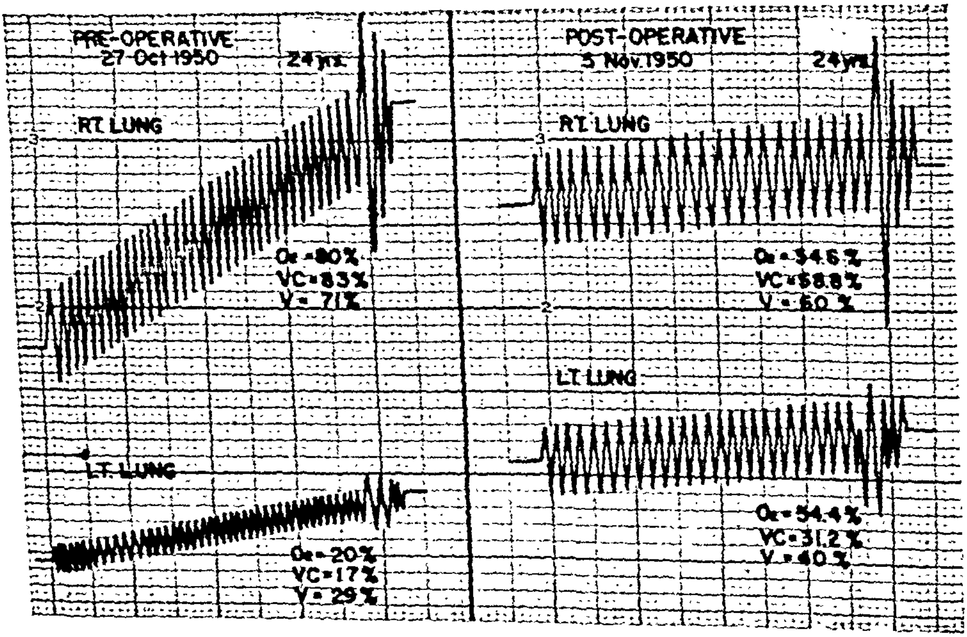

Fig. 8.—(Case 5) Graph of pulmonary function studies.

TABLE IV. BRONCHOSPIROMETRIC DETERMINATION
(VITAL CAPACITY PERCENTAGES)

SIDE INJURED	PREOPERATIVE	POSTOPERATIVE	PERCENTAGE INCREASE CHANGE	PERCENTAGE DECREASE CHANGE
L	31	37.5	7	
R	60	52		8
R	46	58	12	
R	45	45	0	
L	58	56		2
L	37	46.8	10	
L	28	28	0	
L	23	30	7	
L	13	37	24	
R	36.4	53	17	
L	43.3	30		13
R	41	53	12	
L	26.5	20		7
L	20	25	5	
R	35.2	37.5	2	
R	74.5	80	6	
L	17	31.2	14	

left lung, and the left diaphragm was elevated and fixed. Decortication and removal of one foreign body in the left lung was accomplished on Nov. 3, 1950. Bronchoscopy was negative. Pulmonary function determination thirty-four days postoperatively revealed an increase of four liters in the maximum breathing capacity. On the operated side the oxygen consumption increased 34 per cent, vital capacity 14 per cent, and ventilation 11 per cent (Fig. 8).

The correlation of the factors of oxygen, vital capacity, and ventilation pre- and postoperative is not attempted, but it is interesting to note that only two patients showed a decrease in ventilation postoperatively on the injured side, and this was slight (5 per cent and 6 per cent). This may be partially due to the fact that these patients had apparently normal lungs before injury, their youth, and that the lung was encased for a relatively short period. Re-expansion permits but it does not necessarily follow that ventilation will be increased, and the over-all percentage increase of the sixteen patients was only 9 per cent (Table II). Vital capacity increase averaging 11 per cent occurred on the operated side in eleven patients, while four showed a decrease of 8 per cent, and in two there was no change (Table IV). This may be due to the relatively early period after operation that the determinations were made, and further increase may develop later. Physiotherapy was a constant adjunct in therapy. The increase in oxygen consumption on the operated side averaged 17 per cent in fourteen patients, and a decrease averaging 9 per cent occurred in five patients (Table III).

SUMMARY

This presentation reports the findings pre- and postoperatively of certain pulmonary function tests in 19 unselected patients suffering from hemothorax due to war injuries and treated by pulmonary decortication. These determinations were made early following operation, averaging sixty days, and studies made after a longer postoperative interval might alter these findings. These observations indicate that the most uniform finding is an improvement in the percentage of ventilation on the operated side, that oxygen consumption, vital capacity, and maximum breathing capacity show a trend toward restoration of normal function on the injured side, and finally that in individual instances the findings are often unpredictable.

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LATE CHANGES IN VENTILATORY FUNCTION FOLLOWING THORACOPLASTY

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SEVERAL patients have been observed in our follow-up clinic with excellent therapeutic results after thoracoplasty, but complaining of increasing respiratory distress. They all demonstrated evidence of a rotoscoliosis frequently with prominence of the seventh cervical transverse process. Since scoliosis usually is a progressive deformity, the present study was undertaken to determine the changes in ventilatory function which occurred, and to attempt to analyze some of the factors which led to these changes.

Cournand[1, 2] and his co-workers made a detailed study of the early changes in pulmonary function following thoracoplasty. They concluded that the reduction in maximum breathing capacity following a standard seven rib procedure as a rule was not greater than 15 per cent. Furthermore, they observed that when scoliosis developed, the maximum breathing capacity was further reduced. This resulted in an increased tendency to dyspnea on mild exercise. More recently Gaensler and Strieder[3] have published data indicating that the reduction in maximum breathing capacity six months after thoracoplasty was greater (about 35 per cent) in patients in whom the lung, to be collapsed later, contributed 40 per cent or more of the oxygen intake. No mention is made of the number of ribs resected.

The subjects for this investigation were a group of twenty-four patients who had undergone a thoracoplasty three to twenty-seven years before; eight had been studied preoperatively. The only criterion of selection was that sputum had become negative following operation and had remained so up to the time of study.

METHODS

The data were obtained on an outpatient basis on individuals who had been asked to omit breakfast and had been allowed to rest for about thirty minutes before the studies were begun. Vital capacity and maximum breathing capacity were measured on a Benedict-Roth spirometer with ventilometer attachment. Predicted values were calculated from the formulas of Baldwin, Cournand, and Richards.[4] A study of serial roentgenograms on each patient was also made, with particular attention to the presence and development of scoliosis, the length of the anterior segments of the second, third, and fourth ribs removed, and the excision of the transverse processes.

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TABLE I. EIGHT CASES STUDIED BEFORE AND AFTER THORACOPLASTY

CASE	YEARS POST-OPERATIVE	NO. OF RIBS	ANTERIOR SEGMENTS 2, 3, 4	TRANS-VERSE PROCESS	SCOLIOSIS	VITAL CAPACITY IN LITERS				MAXIMUM BREATHING CAPACITY L./MIN.			
						PRED.	OBS.	% PRED.	% CHG.	PRED.	OBS.	% PRED.	% CHG.
RB	P	9	2, 3	yes	mod. sev.	2.90	1.58	55		100	67	67	67
	6 mo.					2.80	1.00	36	35	100	42	42	37
	3					2.70	1.08	40	33	100	47	47	30
RH	P	9	no	yes	slight	4.70	3.29	70		118	109	92	
	2					4.40	2.57	59	16	124	86	70	24
	9					4.20	2.76	65	7	119	84	72	22
FH	P	7	2, 3, 4	yes	severe	5.20	2.70	52		132	57	43	
	4 mo.					5.20	1.78	34	35	132	38	29	33
	6					5.20	1.75	33	37	132	25	26	40
CV	P	7	2	yes	slight	3.15	1.89	50		116	54	55	
	3 mo.					3.24	1.40	43	14	106	42	40	27
	11 mo.					3.24	1.60	49	2	106	51	48	13
	9					3.50	1.40	40	20	99	46	46	16
WC	P	7	no	no	none	4.60	3.38	74		123	95	78	
	2½					4.90	3.15	65	12	131	100	77	1
MR	P	7	no	none	none	4.20	2.89	69		124	70	56	
	7					4.20	2.95	70	0	124	57	46	18
	8					4.20	2.40	57	17	119	65	52	7
OM	P	3 lt.	no	yes	none	4.10	3.66	89		115	92	80	
	2 mo.					4.10	2.80	68	24	115	79	69	14
	5	5 rt.	no	no	none	4.20	2.80	67	24	111	88	80	0
	5 Lt., 2 mo. rt.					4.10	2.30	56	37	108	94	87	0
JR	P	3 rt. 5 lt.	no 2, 3	no no	none none	4.88	2.46	51		111	62	56	
	3 mo.					4.88	2.48	51	0	111	57	51	9
	9					4.88	2.58	51	0	111	55	49	12

TABLE II. TWENTY-FOUR CASES STUDIED AFTER THORACOPLASTY

CASE	YEARS POSTOPERATIVE	NO. OF RIBS	ANTERIOR SEGMENTS 2, 3, 4	TRANSVERSE PROCESS	SCOLIOSIS	VITAL CAPACITY IN LITERS			MAXIMUM BREATHING CAPACITY L./M.		
						PRED.	OBS.	% PRED.	PRED.	OBS.	% PRED.
MK	15	11	2, 3, 4	no	severe	2.70	1.00	36	98	21	22
LB	21	11	2, 3, 4	no	moderate	3.98	1.80	45	95	40	42
GH	9	10	2, 3, 4	yes	severe	3.08	1.72	56	72.5	43	59
H	17	10	no	no	none	4.82	3.36	70	138	125	90
CL	8	9	2	yes	none	3.00	1.85	62	80	73	90
HE	5	9	2, 3, 4	yes	severe	3.18	1.10	34	77	59	75
RB	3	9	2, 3	yes	mod. sev.	2.70	1.08	40	100	47	47
RH	9	9	no	yes	slight	4.20	2.76	65	110	84	72
SP	27	9	no	no	none	2.82	2.56	90	81	81	100
TD	3	8	no	yes	none	3.90	2.70	70	104	49	47
FH	6	7	2, 3, 4	yes	severe	5.20	1.75	33	132	25	19
GN	3	7	2, 3, 4	yes	severe	4.30	2.25	52	104	45	43
GI	5	7	2, 3, 4	yes	mod. sev.	5.20	2.02	39	145	68	47
MI	5	7	2, 3, 4	no	mod. sev.	2.85	1.54	54	97	45	48
SD	3½	7	2, 3, 4	no	mod. sev.	3.70	1.90	50	115	58	50
SR	22	7	2, 3, 4	no	mod. sev.	4.40	2.20	50	123	78	63
GV	9	7	2	yes	slight	3.50	1.40	40	99	46	46
TA	5	7	no	no	diaphragm fixed	3.21	1.15	36	89.1	43	48
MR	7	7	no	no	none	4.20	2.40	57	110	65	52
WC	2½	7	no	no	none	4.90	3.15	65	131	100	77
KI	6	7	no	no	none	4.00	3.30	82	113	97	91
LM	6	7	no	no	none	3.25	2.21	68	84	84	100
JR	9	3 rt. 5 lt.	2, 3	no	none	4.88	2.58	51	111	55	49
OM	5 2 mo.	3 lt. 5 rt.	no	yes	none	4.10	2.30	56	108	94	87

RESULTS

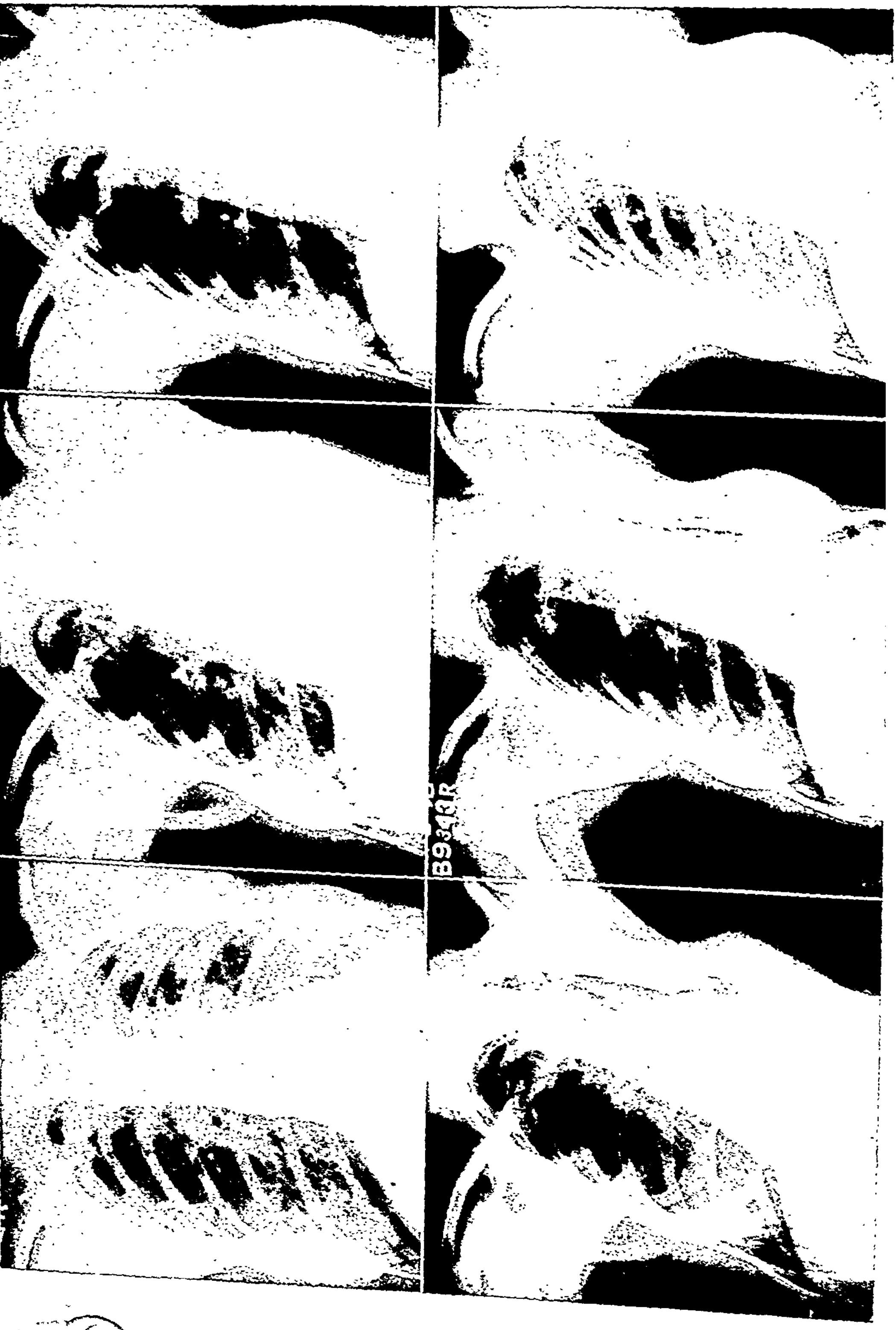
In the tables the cases have been grouped according to the number of ribs resected. Table I shows the results in those patients with preoperative and follow-up studies; Table II, the last postoperative result in each case. It will be seen that frequently the number of ribs resected was not the predominant factor in determining the functional result, and also the individuals' preoperative ventilatory function determined, to a large extent, the postoperative result. Of the twenty-four cases studied, eleven had at least a moderate degree of scoliosis. The maximum breathing capacity in this group averaged 46 per cent of predicted. In two patients studied pre- and postoperatively who developed scoliosis, the change in maximum breathing capacity was over 30 per cent. It also can be seen from the tables that the presence of scoliosis was invariably accompanied by a significant loss in ventilatory function. In addition, the removal of the first rib along with long anterior segments of the second, third, and fourth ribs and the transverse processes was usually associated with the appearance of the deformity. In those cases where one of these structures was left in place, the preservation of long anterior segments seemed most important in limiting the deformity. Although there were some patients without scoliosis in whom the functional result was poor, every case with good ventilatory function had a straight spinal column. It may be noted that when scoliosis did not develop there was slight improvement in late studies when compared to early postoperative figures.

The tables reveal a moderately high incidence of diminished ventilatory function. It is probable that the group with preoperative studies were frequently those in whom a diminished function was suspected, and hence this group is not a representative sample of patients undergoing thoracoplasty. It should be noted that the remainder of the patients in whom scoliosis is not present have figures which do not differ significantly from the predicted.

DISCUSSION

It has been pointed out that with the occurrence of scoliosis there is a considerable loss in function. This fact has been previously reported both in simple thoracoplasty[1, 2] and in thoracoplasty combined with pneumonectomy.[5, 6] Cournand and Berry[5] have reported a case after pneumonectomy (W. J.) in whom a progressive loss in ventilatory function due to overdistention was temporarily corrected by a thoracoplasty, but following the appearance of a severe, progressive scoliosis, much of the improvement was lost.

In most cases where long anterior segments of the second, third, and fourth ribs were removed, along with the transverse processes, a scoliosis of more than moderate degree resulted. Occasionally this was progressive over an extended period (Fig. 1). The occurrence of such a deformity when these structures were left in place was rare. Since there are many other factors which determine the functional result, the absence of a scoliosis does not insure good function. However, in every case where a significant degree of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scoliosis did occur, the result from a functional standpoint was poor. A possible explanation for the deformity may be found by considering the muscular support of the thorax. There are four groups of muscles which act in opposition on the two hemithoraces; the paraspinals, the scalenes, the pectoralis minor, and the serratus. The complete removal of the first and second rib detaches the scalenes: the excision of the transverse processes, the paraspinals; and the removal of the long anterior segments of the second, third, and fourth ribs, the pectoralis minor. In freeing the scapula, the upper digitations of the serratus are often likewise divided. The muscles of the opposite side are now unopposed, and hence may undergo contracture with a resulting rotoscoliosis. It has been shown that if some of the above-mentioned structures are left in place, the deformity is much less severe. It appears that the most important structure in stabilizing the thorax is the long anterior rib segments. We have observed patients in whom all these structures were removed without scoliosis developing. Some of the factors which would tend to limit the appearance of deformity under these circumstances are: the presence of an empyema with resulting fibrosis of the pleura and fixation of the thoracic cage, the presence of a relatively long time interval between stages with reossification of the ribs, and finally the use of adequate physical therapy. The catching of the tip of the scapula beneath a lower rib has frequently been associated with severe and progressive scoliosis.

This report is not intended as a criticism of the over-all excellent results of thoracoplasty, but rather as a study of one of its complications. In some cases a radical procedure with its anticipated deformity and poor functional result may be the price of an adequate therapeutic result. Since, however, the majority of tuberculous lesions occur in the posterior and apical segments of the lung, it may seem advisable to do a less radical anterior thoracoplasty in these cases. The introduction of improved roentgenogram methods, such as lateral tomography, may, by more accurate localization of the lesions, permit a more limited operation which will still produce an adequate therapeutic result. In these lesions where their location requires the removal of long anterior segments to obtain an adequate collapse, it is possible that a better functional result could be obtained by combining pulmonary resection with a less radical thoracoplasty.

SUMMARY

1. Evidence is presented to show that the development of scoliosis after thoracoplasty resulted in a marked decrease in ventilatory function.

2. Some of the features which contribute to the development of scoliosis are discussed.

We wish to express our thanks to Dr. Andre Cournand for his helpful suggestions.

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LUNG FUNCTION STUDIES IN POUDRAGE TREATMENT OF RECURRENT SPONTANEOUS PNEUMOTHORAX

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IT IS now generally recognized that recurrent spontaneous pneumothorax of nontuberculous origin is best handled surgically. Several forms of treatment are available, but exploratory thoracotomy, resection of the pulmonary cysts, blebs, or bullae, with or without additional talcum powder poudrage to induce pleuritis, seems to be the preferred method of treatment. The use of talc to induce pleural symphysis has been criticized because of its supposed damage to lung function, and the purpose of this paper is to report on lung function studies in recurrent spontaneous pneumothorax treated by this method.

In the past a variety of irritating substances have been placed in the pleural cavity to cause pleuritis of sufficient degree to obliterate the pleural space. These substances have been inserted by needle, thoracoscope, or open thoracotomy. For example, Spengler, in 1906, utilized silver nitrate and later a solution of dextrose. Apostolides first used Guaicol and later suggested iodoform. Chandler preferred Gomerol. Iodized oil was introduced by Hennell and Steinberg, and iodized talc by Bethune and Medici.

The introduction of techniques of bronchospirometry made possible the study of individual lung function. The oxygen consumption and the ventilation of the individual lungs were found to be measurements of prime importance. The right lung being normally larger than the left utilizes from 52 to 56 per cent of the total oxygen consumed and does approximately the same proportion of the total ventilation. Pinner and his associates used bronchospirometric techniques in a series of patients and analyzed those cases in which a lung used less than 25 per cent of the total oxygen consumption. These authors found that most of their patients with markedly decreased lung function had pleural rather than parenchymal lesions. They stated, "Many lungs with active parenchymal lesions without pleural involvement show relatively little functional damage even if parenchymal lesions are extensive. It is of great practical importance that pleural involvement which cannot always be diagnosed by roentgenologic examination may lead to more serious functional damage than do much more obvious parenchymal lesions." Likewise, the statement has been made that pulmonary functional capacity is decreased in direct proportion to the severity of pleural symphysis, and that the production of a chemically induced severe pleuritis is inadvisable. Croxatto and Sampietro in a recent publication stated that minimal trauma to the pleura produced adhesions comparable in consistency to those produced by the instillation of irritating substances and supported their findings by pathologic sections.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

There have been thirty cases of recurrent spontaneous pneumothorax treated by exploratory thoracotomy and talc poudrage during the period 1943 to 1950 at Walter Reed Army Hospital and George Washington University Hospital. The great majority of these patients were in the military service at the time of the operation and all went back to military duty following operation. Long follow-ups have not been possible in all of these cases, but to our knowledge there have been no recurrences. Several of the patients were pilots and all returned to flying status after going back to duty. Four patients were chosen as representative of the group and underwent lung function studies before and after operation. Three of these had suffered repeated left-sided spontaneous pneumothoraces, and one had had several episodes of spontaneous pneumothorax on both the right and left side. Five operations were done on the four patients, and in all but one it was possible to excise the lesion suspected of being responsible for the production of the pneumothorax. This corresponds well with a report by Brewer, Dolley, and Evans in which the lesion was found in fourteen of fifteen of their own cases. These authors further reported that in a review of the literature the lesion was found in eighty-seven of ninety-five cases treated by thoracoscopy or thoracotomy. This would appear to indicate the advisability of exploration in these patients.

Prior to operation each of these patients was instructed in and became thoroughly familiar with Specific Remedial Breathing Exercise. The importance of these breathing exercises was emphasized to the utmost, and their active practice was begun early in the postoperative period. Beginning on the third postoperative day the patient reported to the Physical Medicine Clinic where a trained therapist guided them through exercise periods. The authors attribute much of the rapid complete return of good lung function to the diligent application of these exercises.

CASE REPORTS

CASE 1.—This patient was a 25-year-old white man admitted to Walter Reed Army Hospital following three proved episodes of left spontaneous pneumothorax over a two and one-half year period. A left exploratory thoracotomy revealed a small area of subpleural blebs at the apex of the left upper lobe which was excised. The left upper lobe was lightly powdered with sterile talc, and the chest closed with one posterior drain.

TABLE I

	BEFORE OPERATION			TWO MONTHS AFTER OPERATION		
	BOTH LUNGS	RIGHT LUNG	LEFT LUNG	BOTH LUNGS	RIGHT LUNG	LEFT LUNG
Complemental air	1200	1025	1155	1055	860	660
Tidal volume	590	525	265	1090	845	425
Supplemental air	1350	400	0	1056	245	240
Vital capacity	3140	1950 58%	1420 42%	3100	1950 60%	1325 40%
Respiratory rate	15	18	18	19	17	17
Minute ventilation	10.4 L.	9.2 L. 66%	4.8 L. 34%	15.8 L.	11.3 L. 72%	4.4 L. 28%
Oxygen consumption (c.c. per minutes)	192	155	125	430	220	220
% oxygen consumption		55%	45%		50%	50%

Pulmonary Function Data.—(See Table I.) Bronchospirometry before the operation showed that the right lung had a vital capacity of 1,950 c.c. (58 per cent of the total vital capacity), a minute ventilation of 9.2 liters per minute (66 per cent of the total minute ventilation), and an oxygen consumption of 55 per cent. The left lung had a vital capacity of 1,420 c.c. (42 per cent of the total vital capacity), a minute ventilation of 4.8 liters per minute (34 per cent of the total minute ventilation), and an oxygen consumption of 45 per cent.

Spirometry data obtained two months after the operation showed that the vital capacity had decreased from 3,140 c.c. to 3,100 c.c., or 80 per cent of normal. The minute ventilation and oxygen consumption were both increased. Bronchospirometry two months after operation showed that the right lung had a vital capacity of 1,950 c.c. (60 per cent of the total vital capacity), a minute ventilation of 11.3 liters per minute (72 per cent of the total minute ventilation), and an oxygen consumption of 50 per cent. The left lung was found to have a vital capacity of 1,325 c.c. (40 per cent of the total vital capacity), a minute ventilation of 4.4 liters per minute (28 per cent of the total minute ventilation), and an oxygen consumption of 50 per cent. These data show that the vital capacity fell from 3,140 c.c. before operation to 3,100 c.c. following operation. The relative vital capacities of the two lungs were unchanged by the operation. The relative minute ventilation of the left lung fell slightly after the operation from 34 per cent to 28 per cent. The per cent oxygen consumption of the two lungs was only slightly changed by the operation.

CASE 2.—The patient was a 23-year-old white man admitted to Walter Reed Army Hospital with a proved history of three left spontaneous pneumothoraces and five right spontaneous pneumothoraces over a period slightly in excess of six months. Left thoracotomy revealed subpleural blebs at the apex of the left upper lobe. After excision light poudrage was accomplished, and the chest closed with one drain. Two months later a right thoracotomy revealed blebs at the apex of the right upper lobe. These were also excised, the lung poudraged, and the chest closed with one drain.

TABLE II

	BEFORE OPERATION			TWO MONTHS AFTER LEFT OPERATION		AFTER BILATERAL OPERATION 4½ MONTHS AFTER LEFT SIDE 2½ MONTHS AFTER RIGHT SIDE		
	BOTH LUNGS	RIGHT LUNG	LEFT LUNG	RIGHT LUNG	LEFT LUNG	BOTH LUNGS	RIGHT LUNG	LEFT LUNG
Complemental air	1050	680	525	500	475	450	250	475
Tidal volume	700	820	450	1050	475	1500	1125	500
Supplemental air	1700	700	125	550	200	1050	375	225
Vital capacity	3450	2200 67%	1100 33%	2100 65%	1150 35%	3000	1750 60%	1200 40%
Respiratory rate	10	9	9	10	10	13	21	21
Minute ventilation	8 L.	5.2 L. 65%	2.8 L. 35%	11.5 L. 72%	4.5 L. 28%	20 L.	20 L. 76%	6 L. 24%
Oxygen consumption (c.c. per minute)	260	180	125	210	125	365	135	330
% oxygen consumption		59%	41%	63%	37%		29%	71%

Pulmonary Function Data.—(See Table II.) The pulmonary function data are summarized in Table II. Before operation the vital capacity was 3,450 c.c. The minute ventilation was 8 liters per minute. Bronchospirometry showed that the right lung had a vital capacity of 2,200 c.c. (67 per cent of the total vital capacity), a minute ventilation of 5.2 liters per minute (65 per cent of the total minute ventilation), and an oxygen consumption of 59 per cent. The left lung had a vital capacity of 1,100 c.c. (33 per cent of the total vital capacity), a minute ventilation of 2.8 liters per minute (35 per cent of the total minute ventilation), and an oxygen consumption of 41 per cent.

Approximately two months after the left-sided operation, bronchospirometry was again performed. At this time the right lung had a vital capacity of 2,100 c.c. (65 per cent of the total vital capacity), a minute ventilation of 11.5 liters per minute (72 per cent of the total minute ventilation), and an oxygen consumption of 63 per cent. The left lung had a vital capacity of 1,150 c.c. (35 per cent of the total vital capacity), a minute ventilation of 4.5 liters per minute (28 per cent of the total minute ventilation), and an oxygen consumption of 37 per cent. The data before and after the operation on the left side showed that the vital capacity on the two sides was unaffected by the poudrage procedure. The relative minute ventilation and the per cent oxygen consumption of the left side were slightly decreased.

Spirometry after the operation on the right side revealed a vital capacity of 3 liters (this was 87 per cent of the value before the first operation). The minute ventilation was 20 liters per minute. Bronchospirometry after the operation on the right side showed that the right lung had a vital capacity of 1,750 c.c. (60 per cent of the total vital capacity), a minute ventilation of 20 liters per minute (76 per cent of the total minute ventilation), and an oxygen consumption of 29 per cent. The left side had a vital capacity of 1,200 c.c. (40 per cent of the total vital capacity), a minute ventilation of 6 liters per minute (24 per cent of the total minute ventilation), and an oxygen consumption of 71 per cent.

From the data cited, it is apparent that poudrage on the left had relatively little affect upon the vital capacity. There was slight decrease in the minute ventilation on the left and a slight decrease in the per cent oxygen consumption on the left.

Four and one-half months after the left-sided operation, which was two and one-half months after the right-sided operation, the vital capacity was still 87 *per cent of the* value found before the first operation. The right and left lung vital capacities were not significantly changed by either poudrage. The minute ventilation of the right lung had *increased* from 65 per cent to 76 per cent of the total minute ventilation, whereas the per cent oxygen consumption of the right lung had risen from 59 per cent before the first *operation* to 63 per cent after the first operation, but then had fallen to 29 per cent after the second operation.

CASE 3.—The patient was a 21-year-old white man who had had three episodes of left spontaneous pneumothorax within three months. Left exploratory thoracotomy revealed tiny, subpleural blebs covering the entire lung surface. No leakage was apparent at operation, and no excision was carried out. The entire left lung was lightly powdered and the chest closed with drainage.

TABLE III

	BEFORE OPERATION			THREE MONTHS AFTER OPERATION		
	BOTH LUNGS	RIGHT LUNG	LEFT LUNG	BOTH LUNGS	RIGHT LUNG	LEFT LUNG
Complemental air		625	340	875	510	370
Tidal volume		520	240	650	300	240
Supplemental air		280	170	825	340	240
Vital capacity	2700	1425	750	2350	1150	850
		66%	34%		58%	42%
Respiratory rate		16	16	23	30	30
Minute ventilation		4.5 L.	1.8 L.	16.7 L.	8.4 L.	4.6 L.
		71%	29%		65%	35%
Oxygen consumption (c.c. per minute)		365	65	208	175	140
% oxygen consumption		85%	15%		56%	44%

Pulmonary Function Data.—(See Table III). Before operation this patient's vital capacity was 2,700 cubic centimeters. Bronchospirometry revealed that the vital capacity of the right lung was 1,425 c.c. (66 per cent of the total vital capacity), the minute ventilation was 4.5 liters per minute (71 per cent of the total minute ventilation), and the oxygen consumption was 85 per cent. The vital capacity of the left lung was 750 c.c. (34 per cent of the total vital capacity), the minute ventilation was 1.8 liters per minute (29 per cent of the total minute ventilation), and the oxygen consumption was 15 per cent.

Spirometry approximately four months after the operation on the left side showed that the vital capacity was 2,350 c.c., and the minute ventilation was 16.7 liters per minute. Bronchospirometry nearly three months after the operation showed that the right lung had a vital capacity of 1,150 c.c. (58 per cent of the total vital capacity), the minute ventilation was 8.4 liters per minute (65 per cent of the total minute ventilation), and the oxygen consumption was 56 per cent. The left lung had a vital capacity of 850 c.c. (42 per cent of the total vital capacity), a minute ventilation of 4.6 liters per minute (35 per cent of the total minute ventilation), and an oxygen consumption of 44 per cent.

The left poudrage had little effect upon the total vital capacity but tended to increase the vital capacity of the left side. The minute ventilations of the two sides were not significantly changed, but again the left side showed a relative increase. However, the per cent oxygen consumption of the left side showed a distinct increase from 15 per cent before the operation to 44 per cent after the operation.

CASE 4.—The patient was a 20-year-old white man who suffered a right spontaneous pneumothorax one and one-half years prior to admission without recurrence. During that period three left spontaneous pneumothoraces occurred. At left thoracotomy apical subpleural blebs were seen and excised. The left upper lobe was lightly powdered and the chest was closed with drainage.

TABLE IV.

	BEFORE OPERATION			TWO MONTHS AFTER OPERATION		
	BOTH LUNGS	RIGHT LUNG	LEFT LUNG	BOTH LUNGS	RIGHT LUNG	LEFT LUNG
Complemental air		1150	1000	1585	1280	935
Tidal volume		400	100	1325	340	245
Supplemental air		250	0	1400	480	245
Vital capacity	4400	1800	1100	4290	2100	1325
		62%	38%		61%	39%
Respiratory rate		28	28	17	26	26
Minute ventilation		8.5 L.	1.4 L.	18.8 L.	6 L.	3 L.
		86%	14%		67%	33%
Oxygen consumption (c.c. per minute)		365	135	360	350	130
% oxygen consumption		73%	27%		73%	27%

Pulmonary Function Data.—(See Table IV.) This patient's vital capacity before operation was 4,400 c.c. Bronchospirometry before operation showed that the right lung had a vital capacity of 1,800 c.c. (62 per cent of the total vital capacity), a minute ventilation of 8.5 liters per minute (86 per cent of the total minute ventilation), and an oxygen consumption of 73 per cent. The left lung was found to have a vital capacity of 1,100 c.c. (38 per cent of the total vital capacity), a minute ventilation of 1.4 liters per minute (14 per cent of the total minute ventilation), and an oxygen consumption of 27 per cent.

Two months after operation spirometry data revealed that the vital capacity was 4,290 c.c. Bronchospirometry two months after operation revealed that the right lung had a vital capacity of 2,100 c.c. (61 per cent of the total vital capacity), a minute ventilation of 6 liters per minute (67 per cent of the total minute ventilation), and an oxygen consumption of 73 per cent. The left lung was found to have a vital capacity of 1,325 c.c. (39 per cent of the total vital capacity), a minute ventilation of 3 liters per minute (33 per cent of the total minute ventilation), and an oxygen consumption of 27 per cent.

These data show that the operation caused a 3 per cent decrease in vital capacity from 4,400 c.c. to 4,290 c.c. The relative vital capacities of the right and left lung were unchanged. The relative minute ventilation of the left lung increased from 14 per cent before the operation to 33 per cent after the operation. The per cent oxygen consumption of the two lungs was unchanged by the operation.

TABLE V. SUMMARY OF PULMONARY FUNCTION CHANGES CAUSED BY POUDRAGED

	VITAL CAPACITY BOTH LUNGS	% VITAL CAPACITY		% MINUTE VENTILATION		% OXYGEN CONSUMPTION	
		RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
Patient 1	3% ↓	2% ↑	2% ↓	6% ↑	6% ↓	5% ↓	5% ↑
Patient 2		2% ↓	2% ↑	7% ↑	7% ↓	4% ↑	4% ↓
	13% ↓ Bilateral operation	7% ↓	7% ↑	11% ↑	11% ↓	30% ↓	30% ↑
Patient 3	13% ↓	8% ↓	8% ↑	6% ↓	6% ↑	29% ↓	29% ↑
Patient 4	3% ↓	1% ↓	1% ↑	19% ↓	19% ↑	N.C.	N.C.

SUMMARY

Thirty patients have undergone exploratory thoracotomy and talcum powder poudrage in the treatment of recurrent spontaneous pneumothorax. Pulmonary function studies were accomplished on four patients, one of whom had surgery performed on both sides.

In Case 1 the vital capacity fell 3 per cent after the poudrage. The relative vital capacity of the left lung fell 2 per cent, the relative minute ventilation of the left lung fell 6 per cent, and the per cent oxygen consumption of the left side rose 5 per cent. These changes do not seem significant.

In Case 2 the vital capacity fell 13 per cent after the bilateral operation. After the left operation alone, the relative vital capacity of the left lung rose 2 per cent, the relative minute ventilation of the left lung fell 7 per cent, and the per cent oxygen consumption of the left side fell 4 per cent. None of these changes seem to be significant. After the second operation on the right side, the relative vital capacity of the right lung fell 7 per cent, the relative minute ventilation of the right lung rose 11 per cent, and the per cent oxygen consumption of the right side fell 30 per cent. The fall in per cent oxygen consumption on the right side after the right poudrage seems significant.

In Case 3 the vital capacity fell 13 per cent after the poudrage. The relative vital capacity of the left lung rose 8 per cent, the relative minute ventilation of the left lung rose 6 per cent, and the per cent oxygen consumption of the left side rose 29 per cent. This increase in oxygen consumption on the left side seems to be significant.

In Case 4 the vital capacity fell 3 per cent after the poudrage. The relative vital capacity of the left lung rose 1 per cent, the relative minute ventilation of the left lung rose 19 per cent, and the per cent oxygen consumption of the left side was unchanged. This increase in minute ventilation on the left side seems significant.

CONCLUSIONS

Open thoracotomy and talcum powder poudrage for recurrent spontaneous pneumothorax produced no consistent decrease in pulmonary function as determined by bronchospirometry in four patients. The observed changes in pulmonary function do not seem to be of greater degree than would be expected to follow any open thoracotomy. We conclude that the introduction of talcum powder to induce pleural symphysis has no demonstrable harmful effects during the period of our observation.

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DISCUSSION ON "PROGRESSIVE CHANGES OF PULMONARY FUNCTION AFTER PNEUMONECTOMY: THE INFLUENCE OF THORACOPLASTY, PNEUMOTHORAX, OLEOTHORAX, AND PLASTIC SPONGE PLOMBAGE ON THE SIDE OF THE PNEUMONECTOMY," BY DR. EDWARD A. GAENSLER, AND DR. JOHN W. STREIDER; "PULMONARY FUNCTION IN TRAUMATIC HEMOTHORAX TREATED BY DECORTICATION," BY DR. JAMES H. FORSEE AND MAJOR STEPHAN L. KYLAR; "LATE CHANGES IN VENTILATORY FUNCTION FOLLOWING THORACOPLASTY," BY DR. S. R. POWERS, JR., AND A. HIMMELSTEIN; AND "LUNG FUNCTION STUDIES IN POUDRAGE TREATMENT OF RECURRENT SPONTANEOUS PNEUMOTHORAX," LT. COLONEL JOHN PAUL, DR. EDWARD J. BEATTIE, JR., AND DR. BRIAN BLADES.

DR. FELIX A. HUGHES, Jr., Memphis.—I should like to discuss the paper on idiopathic spontaneous pneumothorax. We have had fifty-nine patients with spontaneous pneumothorax, amounting to 3.1 per cent of admissions to the thoracic surgery section at Kennedy Hospital. Observation and aspiration were sufficient treatments in twelve cases with small or minimal collapse. Major thoracotomy was used in five patients, because of large bullae in four patients and a clotted hemothorax in one.

Forty-two patients have been treated by closed drainage, using a Foley catheter with satisfactory results in forty of the forty-two. Expansion was prevented by a membrane over the lung of one patient. Although the lung expanded, large bullae required removal in the other case. Recurrence of pneumothorax occurred in three of the forty cases in which we had satisfactory results, and the lung re-expanded with the use of aspiration or drainage.

If poudrage will prevent recurrence, if it will allow maximum pulmonary function, if it is free of danger as a foreign body, its use might be advocated. I think if poudrage is not used the dangers of aspiration and of rapid expansion of the lung have been greatly exaggerated. Timidity and masterful inactivity are usually out of place in the treatment of spontaneous pneumothorax.

I would like to stress two points: First, closed drainage should usually be initiated early since re-expansion is often obtained in a matter of twenty minutes, even when the patient has been treated conservatively for months. Closed drainage is a ward procedure and offers little more discomfort than a thoracentesis. There is no immobility of the patient, since he can move around with a bottle or flask attached for drainage.

(Slides.) This patient had a pneumothorax and was treated conservatively for five months.

(Slide.) This shows the patient as he was discharged five days later.

The second thing I would like to stress is the use of a Foley or balloon catheter.

(Slide.) With the balloon inflated, the catheter tip can be held safely close to the chest wall. A catheter inserted 12 inches or more with the tip under thick fluid or fibrin cannot be expected to cause the lung to expand satisfactorily.

(Slide.) This shows adhesions and an air pocket at the site of the balloon and also a slight pneumothorax. It might be better to deflate the balloon a few hours before the catheter is removed.

This type of treatment of spontaneous pneumothorax has given satisfactory results in a high percentage of cases within a very short time.

DR. EARLE B. KAY, Cleveland.—I would like to discuss the paper by Dr. Gaensler and Dr. Strieder relative to postoperative pulmonary distention. The objective of pulmonary surgery is the removal of diseased tissue and the preservation of function of the residual lung. It was noted with concern that a certain number of patients developed basilar distention in the residual lobe following a previous lobectomy to an unfavorable degree in order to occupy the space previously occupied by the resected lobe. In an effort to determine the fate of the residual lobe following previous lobectomy function studies, bronchospirometric determinations, roentgenograms, and bronchograms were performed on a group of thirty-three postoperative patients followed from seven to seventy-three months, with an average of forty-one months. The pulmonary function studies were performed by Drs. Haven Perkins and Philip Crastnopol in the Respiratory Laboratory at Crile and Brecksville Veterans Hospitals. From these studies it was of interest to note the apparent value derived from performing a temporary phrenic neuremphraxis early in the postoperative period as a space reducing procedure. The patients not having phrenic operations (twenty-one) were compared to those who had the procedure (twelve).

(Slide.) Of twenty-one patients who did not have phrenic operations, thirteen (62 per cent) had either no or only minimal basilar distention. Eight of the patients (38 per cent) had moderate basilar distention. In contrast, in eleven of the twelve patients having associated phrenic operations, or 92 per cent, there was either only minimal (three cases) or no evidence of basilar distention (eight cases).

(Slide.) The ability of the residual lobe on the operated side to ventilate was much better in the phrenic group (75 per cent had normal ventilation) as compared to the nonphrenic group (33 per cent with normal ventilation).

(Slide.) The oxygen consumption in the nonphrenic group was below normal in 66 per cent of the patients, and in the phrenic group in only 26 per cent of the patients.

(Slide.) The ventilatory equivalent was within normal limits in 24 per cent of the patients without phrenics on the operated side as compared to 58 per cent within normal limits of those with phrenics.

From these studies it was felt that a temporary phrenic neuremphraxis performed early in the postlobectomy period was of value in preserving pulmonary function.

DR. G. W. WRIGHT, Saranac Lake, N. Y.—I would like to comment on three of the four papers. In regard to Dr. Gaensler's paper, I think we will all agree that to subject a patient to a space-correcting operation is a fairly serious procedure, and one should take every pains to attempt to justify this action. The question of what overdistention means seems to me to be very important. For the majority of any twenty-four hour period of the day we do not go around with our lungs in a state of full expiration which is the size of the lung with residual air in it. Nor do we go around with our lungs maximally full, which is the size of the lung expressed by the total volume. *Actually, for the majority of the* twenty-four hour day the functional residual air determines the size or state of distention of the lung. In looking at Dr. Gaensler's data, it is perfectly obvious that the functional residual air is enlarged to some extent after resection without subsequent thoracoplasty or plombage. The important feature to me, however, is—what is the degree of this enlargement—and I would presume from looking at his data that it was of the order of a 15 to 20 per cent increase, some 300 c.c. which in terms of actual stretch of the lung causes only about 1 or 2 cm. of water change in the intrapleural pressure. In other words, I have the feeling that from his data the degree of distention is not very great from the actual physical point of view. Certainly it is not as great as happens in all of us when we change from the recumbent position to the standing position. As you know the functional residual air is increased by from 50 to 100 per cent in the normal man by a change from the recumbent to the standing position.

In view of the very slight effect of pneumonectomy on maximum breathing capacity, I think one should question whether or not there is clear-cut physiologic evidence to warrant doing a space-correcting operation after lobectomy, and perhaps even after pneumonectomy. I would like to ask Dr. Gaensler whether or not he believes his data indicate that the doing of a space-correcting operation is warranted on a functional basis in people after pneumonectomy.

In regard to the interesting patient reported by Dr. Forsee, I would like to point out that it is one thing to talk about pleural damage influencing lung function when the damage is caused by empyema; it is quite another when one speaks only of pleural synthesis. It is well known that pleural synthesis does not influence appreciably pulmonary function; for example all birds and certain mammals have no pleural space—the elephant has no pleural space. These animals do very well, and many human beings do very well with synthesis of the pleura. Therefore I would think that if a simple synthesis of the pleura may be accomplished, it would be a very useful thing in recurrent spontaneous pneumothorax.

Dr. Powers' paper interested me a great deal because we have wondered about this scoliosis. I did not quite clearly perceive how he distinguished between the functional effects of thoracoplasty per se; that is collapse of lung, and the effects of the torsion due to scoliosis.

DR. STRIEDER (closing).—With regard to Dr. Wright's remarks concerning the indications for a space-correcting procedure following pneumonectomy, I have nothing to say from a physiologic point of view beyond this: Dr. Gaensler's data would seem to indicate to us that there is justification for the procedure. Only summary data were shown on the slides, but details will appear in the published paper. It should then be evident that extreme overdistention may occur early or late, and there is no way of predicting when it will occur in a given case. With this in mind, we believe that means should be taken relatively early to prevent this, since such measures taken late are not so effective.

However, again, as was indicated in Dr. Gaensler's introduction, it is not only on the basis of physiologic data, without wishing to evade the issue that Dr. Wright raises, but there are also clinical facts that lead us under certain circumstances to correct the defect which has arisen as a result of pneumonectomy in a given patient. In this connection I refer to the fact that a patient may be uncomfortable purely on the basis of the mediastinal shift or tracheal deviation, in addition to what appears to us to be clinical evidence of overdistention, namely, that the patient is unhappy and dyspneic on exertion and has ineffectual cough.

DR. HIMMELSTEIN (closing).—Dr. Blalock, may I make one comment about the patient presented by Lt. Colonel Paul before I close?

We have been impressed by the development of talc granuloma in some of the abdominal surgery we have done and have not liked it. In treating recurrent spontaneous pneumothorax, we follow Mr. Brock's procedure, using one-half cubic centimeter of 10 per cent silver nitrate.

The comment is the fact that in these patients at six months we have tried to induce a pneumothorax by the technique usually employed in six or seven spots around the chest, and in six of the seven patients have been unable to find a pneumothorax space.

In closing our paper I would like to answer Dr. Wright's question. We have not specifically listed the effects of thoracoplasty, but merely have pointed out that we have obtained our best results when the spine is straight, and that when scoliosis did occur it usually was accompanied by about 50 per cent or greater reduction in maximum breathing capacity, which is an effect one would not anticipate just from the thoracoplasty.

LT. COLONEL PAUL (closing).—I wish to thank the discussors of the paper. In answer to Dr. Hughes' question concerning chronic pneumothorax, I would state that the present series of cases contained no cases of chronic pneumothorax but was confined to the treatment of recurrent spontaneous pneumothorax. All the patients had experienced at least three episodes of spontaneous pneumothorax of nontuberculous origin. In the military service the problem exists as to whether a man is fit or unfit for full military duty. We have found that thoracotomy with poudrage is a very effective method of handling recurrent spontaneous pneumothorax enabling us to return these patients to a full duty status after a minimum of hospitalization.

THE SIGNIFICANCE OF CELL TYPES IN BRONCHOGENIC CARCINOMA*

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BRONCHOGENIC carcinoma is no longer a disease seen as a curiosity clinically and at necropsy. It is a disease which, if seen early enough, can be eradicated surgically. It is important, therefore, to establish morphologic criteria by which one can determine to some degree operability and chances of survival after resection. Grading bronchogenic carcinoma furnishes little of value surgically or prognostically.

Bronchogenic carcinoma is a primary carcinoma of the lung, of varied histologic types, apparently arising from the surface mucosa of the bronchi or bronchioles. The mucous and serous glands of the bronchial wall are believed to be the site of origin of an entirely different type of tumor, namely, those of the adenoma and cylindroma group. The biologic behavior of tumors of the last-mentioned group is sufficiently unlike that of bronchogenic carcinoma to warrant their exclusion. The definitive diagnosis of bronchogenic carcinoma is not always possible from the examination of a resected specimen, particularly in peripherally placed tumors, because of the simulation of bronchogenic carcinoma and metastatic carcinoma.

In studies to be reported on in the following four papers adenomas and cylindromas were eliminated on the basis of histologic examination of the tumor. In order to exclude metastatic carcinoma, any patient who had a primary carcinoma, elsewhere was excluded from the study.

The histopathologic portion of these studies was made separately from the clinical portion. When the pathologic studies were completed, the clinical, surgical, and cytologic aspects of the various cases were correlated with the various histopathologic groups. The following four papers present the results.

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*Editor: This is an introductory statement to precede four papers: one by McBurney and co-workers, one by Carlisle and co-workers, and two by Patton and co-workers.

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BRONCHOGENIC SMALL-CELL CARCINOMA[*]

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THERE have been many studies of bronchogenic carcinoma, but infrequent ones of individual cell types. Our study is concerned with a clinical and pathologic study of small-cell bronchogenic carcinomas seen at the Mayo Clinic.

METHODS AND MATERIAL

The records and tissue of all cases of tumor of the lung encountered at the clinic through 1948, on which pathologic material was available for study, were reviewed. No cases were included in which the diagnosis was made on a clinical basis only. This involved study of biopsy and cytologic material in 1,153 cases.

It was deemed necessary to restudy all tissue, because in past years the pathologic diagnosis of bronchogenic carcinoma was not broken down according to cell types, and also because we wished to be certain that adenomas or other misdiagnosed lesions were not included. Cases in which there was a roentgenographic shadow in the lung and in which positive results were obtained on biopsy of the cervical nodes were not included because it was felt that this evidence, while suggestive, was not conclusive for carcinoma of the lung.

New sections were cut and stained with hematoxylin and eosin in instances in which the routine slides were unsatisfactory for study. The lungs in all cases in which resection was done were also studied.

Following the study of the microscopic sections the clinical records were then reviewed and follow-up studies done.

CLINICAL DATA

Of the 1,153 cases, 849 were felt to represent primary bronchogenic carcinoma. Of this number, in seventy-five (8.8 per cent) the diagnosis was small-cell bronchogenic carcinoma. An additional fifteen cases were included in the study on the basis of positive cytologic evidence alone.

Ninety per cent of the cases fell into the age group 40 to 69 years (Table I). The youngest patient was 30 years old, while the oldest was 75 years of age.

There were 87 males and 3 females, or a ratio of 29 to 1. Eighty-one of the patients were Anglo-Saxon or Scandinavian, seven Jewish, one Mexican, and one Spanish.

Data on the use of tobacco were not complete enough to be significant. As might be expected, most of the patients were smokers to some degree.

Occupation did not seem to be a significant factor, as there were many occupations represented.

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[*] Abridgement of thesis submitted by Dr. McBurney to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

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TABLE I. AGE DISTRIBUTION IN 90 CASES OF SMALL-CELL BRONCHOGENIC CARCINOMA

AGE, YEARS	CASES	PER CENT
30-39	7	7.8
40-49	20	22.2
50-59	43	47.8
60-69	18	20.0
70 or more	2	2.2
Total	90	100.0

Symptoms and Physical Signs.—The average duration of symptoms was 5.9 months; however, in forty-nine cases the duration was less than four months, with about one-fourth of the patients having symptoms of less than two months' duration (Table II).

TABLE II. DURATION OF SYMPTOMS IN 88 CASES OF SMALL-CELL BRONCHOGENIC CARCINOMA*

DURATION (MONTHS)	CASES	DURATION (MONTHS)	CASES
Less than 1	5	6	3
		7	6
1	20	8	2
2	17	9	3
3	7	10-11	11
4	2	12-23	4
5	5	24	3

*Duration unknown in one additional case. Story unreliable but possibly of three-year duration in one additional case.

The symptoms were identical with those of any group of carcinomas of the lung in general. Table III indicates the early and most prominent symptoms in the cases.

The signs elicited were again those of bronchogenic carcinoma in general. The most significant and striking ones are listed in Table IV. It is of interest that almost 10 per cent of patients had very little evidence of thoracic disease on physical examination.

TABLE III. EARLY SYMPTOMS IN 90 CASES OF SMALL-CELL BRONCHOGENIC CARCINOMA

SYMPTOM	CASES	SYMPTOM	CASES
Cough	58*	Fever	7
Pain in chest	27*	Weakness	6
Dyspnea	16	Vomiting	2
Episodes of pneumonia, influenza or chest colds	15	Anorexia	1
		Abdominal cramps	1
Lost weight	13	Voice lost	1
Hemoptysis	9	Night sweats	1
Wheeze	7	Feeling of something in the chest	1
Fatigue	7	Joint pain or arthritis	6

*In one case this was the only symptom.

Roentgenologic Evidence.—All patients had roentgenologic examination of the chest, but the report on one was not available for this study. The findings in the initial roentgenograms of the chest are listed in Table V.

By roentgenogram the lesion was located on the right in fifty-four cases, on the left in thirty-two, and bilaterally in one. The site was not determined in the remainder.

TABLE IV. SIGNIFICANT PHYSICAL SIGNS IN 90 CASES OF SMALL-CELL BRONCHOGENIC CARCINOMA

SIGN	CASES
Dullness over the affected side	56
Breath sounds diminished or absent over the affected side	45
Lag in respiratory movement over the affected side	16
Râles in the chest over the affected side	11
Palpable supraclavicular or axillary nodes	13
Fixed vocal cord (in two of these cases chest negative on physical examination)	6
Bronchial breath sounds over affected side	3
Evidence of superior vena caval obstruction	4
Emaciation	4
Pigmentation on the trunk, face or extremities	2
Palpable liver	2
Audible stridor	2
Deviated trachea	2
Tenderness of chest wall	2
Cyanosis, palpable abdominal mass, nodule in the abdominal wall, clubbed fingers, fixed diaphragm, weakness of one leg	1 each
Physical examination negative	5
Negative except for:	
Stiff shoulders	1
Loss of weight	1
Small "shotty" cervical nodes	1

TABLE V. INITIAL ROENTGENOGRAPHIC FINDINGS IN EIGHTY-NINE CASES OF SMALL-CELL CARCINOMA OF THE LUNG

FINDINGS	CASES
Hilar mass or perihilar infiltration	34
"Atelectasis" of lobes or part of a lung	26
Diffuse opacity, collapse or infiltration of a whole lung	8
Mass at the base of the lung with or without fixation of the hemidiaphragm	5
Infiltration or mass in the midportion of a lung	7
Mediastinal widening only	5
"Bronchiectasis" only	5
Multiple masses in one lung	2
Multiple cavities with mass at the hilus	2
Single cavity	1
Lesion at the costophrenic angle	2
Esophageal displacement by mediastinal mass	1
Condition obscured by opaque oil	1
Negative except for questionable mediastinal widening	1

Bronchoscopic Findings.—Bronchoscopic examination was made in eighty-one cases. In sixty-seven cases a tumor was seen, but biopsy gave positive results in only sixty-two. Examination revealed some deformity of the bronchi in another seven cases. In one case the findings were those of inflammation of the bronchus, and in five cases bronchoscopy gave completely negative results.

Localization of the lesion as determined bronchoscopically is summarized in Fig. 1.

Cytologic Examination of Sputum and Bronchial Secretions.—Adequate study of the sputum or bronchial secretions had been made in thirty-one of the ninety cases of small-cell carcinoma. By adequate study is meant that at least three different specimens were examined before the result could be called negative.

Of the thirty-one cases, carcinoma cells were present and identified in twenty-nine. This represents a diagnostic accuracy of 93.5 per cent. In fourteen of these twenty-nine cases both cytologic and biopsy material were available for study and of these, in ten the lesion had been diagnosed carcinoma of the small-cell type, and in the remainder it had been diagnosed carcinoma cells, type not specified. Thus, in more than 70 per cent of cases, it was possible to specify cell type as well as diagnose carcinoma.

Treatment.—Since the first successful pneumonectomy for carcinoma by Dr. Evarts Graham was done only in 1933, it is obvious that all the patients in this series could not have had the opportunity of surgical treatment had their lesions been operable at the time. There were, however, only eight cases prior to 1934, so that the majority of the patients could have had surgical treatment if their disease so warranted.

Of the eighty-two patients seen after 1934, only thirty had tumors that were deemed operable, and as one of these refused surgical treatment, there were twenty-nine who were operated on. In other words, about 35 per cent of patients seen were thought clinically to have operable lesions.

Of the twenty-nine patients who underwent thoracotomy, fifteen actually had removal of the lesion, total pneumonectomy being done in every case. In fourteen cases resection was not possible because of extension of the cancer beyond the limits of surgical removal. Details on the duration of life following pneumonectomy in these cases are given in Table VI.

TABLE VI. SMALL-CELL BRONCHOGENIC CARCINOMA: SURVIVAL AFTER PNEUMONECTOMY

SURVIVAL (MONTHS)	PATIENTS	SURVIVAL (MONTHS)	PATIENTS
1	1	12	1
2	1	15	1
3	1	19	1
4	1	26	1
9	4	33	1*
10	1	63	1†

*Operation, January, 1948; alive September, 1950.
†Operation, May, 1945; alive September, 1950.

One patient had bronchoscopic fulguration because of a mistaken diagnosis of adenoma. This patient failed to survive more than six months.

Roentgen Treatment.—Seventeen patients received roentgen treatment. The efficacy of this form of treatment is difficult to evaluate since the effective tumor dose delivered is not known in every case and many patients received palliative or placebo treatment only. None treated with roentgen rays survived longer than a year.

PATHOLOGIC AND CYTOLOGIC ASPECTS OF THE DISEASE

Almost all workers agree on the classification of the cell types, squamous-cell carcinoma and adenocarcinoma, but what to include and exclude in these classifications is not agreed upon. Many group the remainder of the bronchogenic carcinomas as anaplastic or undifferentiated. To illustrate some of the different conceptions regarding classification: Rienhoff[1] considers small-cell (oat-cell) carcinoma, "cylindrical-cell" carcinoma, and adenocarcinoma all as forms of adenocarcinoma, and hence he states that all bronchogenic carcinomas are either adenocarcinomas or squamous-cell carcinomas. Somewhat similarly,

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Ormerod[2] lumps all bronchogenic carcinomas as squamous-cell carcinomas or nonsquamous-cell carcinomas. O'Keefe,[3] Björk,[4] and Graham[5] all state that they find it difficult to separate small-cell carcinomas from adenocarcinomas.

In our study no definite evidence could be found to indicate that small-cell carcinoma is an adenocarcinoma subtype. In some cases perithelial grouping of cells gave a pseudoglandular appearance (Fig. 2*a*), but no true acini or alveolar structures were found in any cases.

This study included all those bronchogenic carcinomas with predominantly small cells, regardless of the particular shape they assumed. Thus, in our opinion, the so-called oat-cell cancer, the small round-cell cancer, and the small spindle-cell cancer or "sarcoma" are all simply variations in form of the same tumor.

Gross Characteristics.—Study of the gross surgical specimens revealed that most of the tumors were large, the largest being 15 cm. in diameter. Three patients had small lesions, measuring 1 by 1 by 1 cm., 1.5 by 1.5 by 1 cm., and 1.2 by 0.8 by 1 cm., respectively.

All specimens demonstrated involvement of the hilar nodes by metastatic cancer.

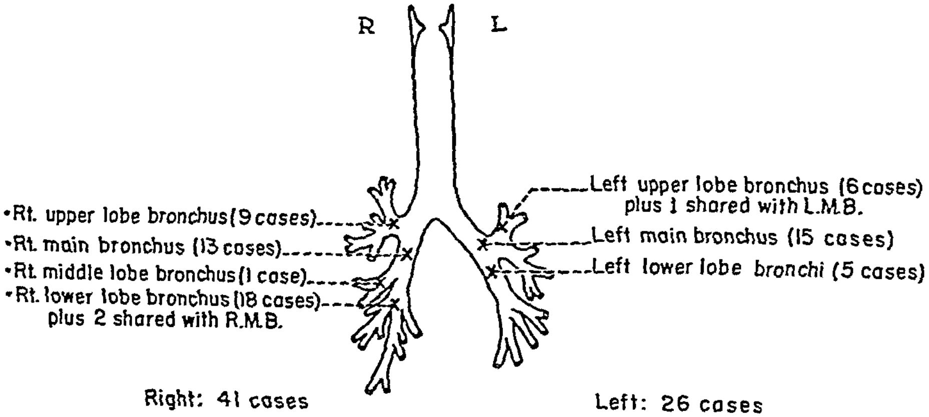
R
L
•Rt. upper lobe bronchus (9 cases)
•Rt. main bronchus (13 cases)
•Rt. middle lobe bronchus (1 case)
•Rt. lower lobe bronchus (18 cases) plus 2 shared with R.M.B.
Left upper lobe bronchus (6 cases) plus 1 shared with L.M.B.
Left main bronchus (15 cases)
Left lower lobe bronchi (5 cases)
Right: 41 cases
Left: 26 cases

Fig. 1.—Small-cell bronchogenic carcinoma. Site of the lesions as determined on bronchoscopic examination.

With only one exception the tumors were located either in the main bronchi or in a major secondary bronchus. The one exception was a 15 by 10 by 8.5 cm. tumor in the peripheral portion of the left lower lobe.

All specimens with one exception had evidence of obstructive pneumonitis distal to the bronchial tumor.

Microscopic Aspects.—From the study of tissue in seventy-five of the ninety cases the following characteristics of this group of tumors were noted.

The most constant feature, of course, was the small size of the cells. To determine the size of these cells and nuclei, measurements of cell size using an eyepiece micrometer were made in twenty cases. In each case the nuclei of at least twenty cells taken at random from representative fields were measured. Because poor definition of the cell membrane is one of the characteristics of this type of tumor a smaller group of actual cell sizes was measured by use of the same method. It was found that the average nuclear diameter was 7.3 microns and the average cell diameter 8.7 microns. Extremes of nuclear size were 3 and 20 microns, but most nuclei fell into the 6- to 8-micron range. For comparison the nucleus of the average lymphocyte measures about 4 microns, and the average polymorphonuclear leucocyte measures about 6 microns (Fig. 2*b*). Thus, compared to many normal cells the malignant cell in question is not small. It is small only in comparison to other malignant cells.

In nearly every case the nuclear-cytoplasmic ratio was high, the nucleus occupying almost 90 per cent of the cell (Fig. 3*a*).

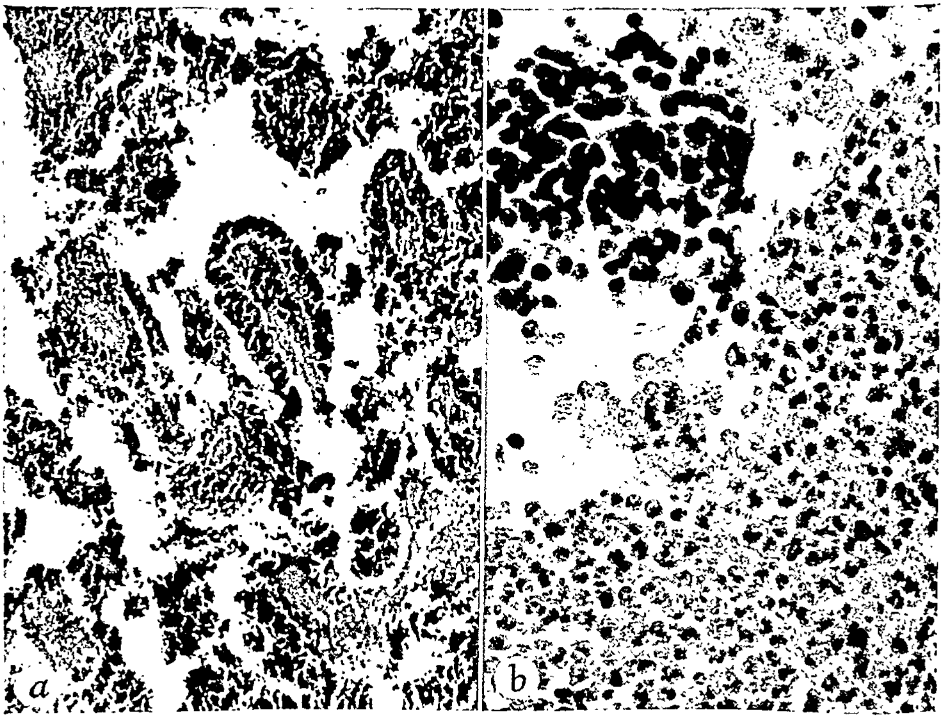

Fig. 2.—Small-cell bronchogenic carcinoma. *a*, Perithelial grouping of cells to give a pseudoglandular appearance (hematoxylin and eosin ×100). *b*, Size of cells of an inflammatory exudate compared with the size of small bronchogenic cancer cells (hematoxylin and eosin ×400).

Hyperchromatism of the nuclei was a prominent feature. In thin sections lightly stained, one could often detect several nucleoli in the nucleus with many fine, scattered, chromatin particles. Most often in the hematoxylin and eosin sections, the nuclei were a solid mass of chromatin. Often one saw only a blob of chromatin material, sometimes spindle-shaped, without any accurate cell or nuclear outlines (Fig. 3*b*). Pleomorphism, giant cells, and very minute cells were present occasionally.

As a rule the cells are closely packed in a medullary form. There is little intercellular stroma nor, as Boyd[6] showed in his cases, do they have intercellular reticulum.

Necrosis forms a very prominent feature of these tumors. This is rarely found in adenomas or lymphosarcomas, and helps in differential diagnosis. Invasion of vessels, lymphatics and adjacent pulmonary tissue and bronchial mucous membrane all were fairly constant features.

Diagnosis of small-cell carcinoma by cytologic examination is based on the finding of numerous cells with a high incidence of more than one of the following[7]: nuclear hyperchromatism, pleomorphism, coarse nucleoli or chromatin granules and a high nuclear-cytoplasmic ratio. Size of cells and nuclei should conform to the average range mentioned. No attempt should be made to diagnose small-cell carcinoma on the basis of a small number of cells, as small histiocytes, lymphocytes, monocytes, and other cells may resemble cancer cells.

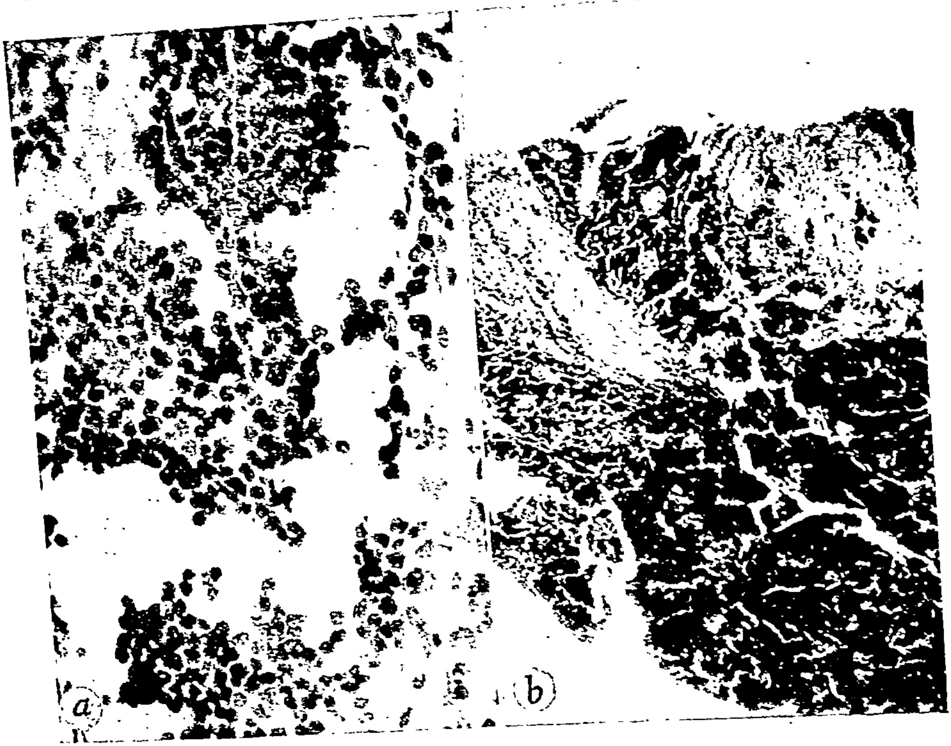

Fig. 3.—Small-cell bronchogenic carcinoma. *a*, High nuclear cytoplasmic ratio with poor outlines of cell membranes (hematoxylin and eosin ×320). *b*, Amorphic mass of chromatin material with poor cell outlines, characteristic of small-cell bronchogenic carcinoma.

COMMENT

Contrary to the belief of Neuhof and Aufses,[8] Tuttle and Womack,[9] Phillips, Basinger, and Adams[10] and also Björk,[4] cell type is a significant prognostic factor and specifically the small-cell carcinoma of the lung is a highly malignant cancer with a poor prognosis, according to the results of our study. Only two survivors, one of whom has had known metastasis, out of ninety cases in forty years' experience, indicates that when this diagnosis is made the outlook is grave indeed.

It is believed that studies of the other cell types of cancer of the lung when correlated with the clinical histories and with this study will indicate that cell type, apart from other clinical factors, will prove to be a most valuable prognostic factor. This view is already held by many,[11, 12] Adams[13] calling it the crux of the whole problem.

The duration of symptoms in most cases makes remote the possibility that small-cell carcinoma is a malignant tumor arising from a previously existing adenoma. There is certainly evidence that adenomas may be, or may go on to become, malignant; however, all the malignant adenomas seen at the clinic were studied as a part of this study, and in every case there was a recognizable difference in the histologic picture from that of the small-cell carcinoma. More study of the malignant adenoma is needed. One should not assume that every case in which small cells show pleomorphism and mitotic figures is one of small-cell carcinoma.

Graham[5, 14] has stated that because of the paucity of necropsy cases of adenoma it is his belief that there is a definite possibility that adenomas become small-cell carcinomas. Abbott[15] has agreed with this belief. Rabin and Neuhof,[16] and also Head[17] have disagreed with this and have stated that a history of a lesion of short duration plus the absence of any polypoid appearance to small-cell carcinomas excludes the possibility of their coming from adenomas. In none of the cases in our series were there any areas in the tumors that resembled the carcinoid or cylindroma pattern of adenomas. If small-cell carcinomas come from pre-existing adenomas, one would expect to find some tissue still present which resembles the parent tumor. At least this is the case in most instances in respect to other malignant tumors that arise from pre-existing benign tumors. The most valuable microscopic criterion for differentiating between small-cell carcinoma and adenoma, aside from the cellular arrangement, is the nuclear-cytoplasmic ratio which is high in small-cell carcinoma and is about 1 to 1 in adenoma. Another feature that aids is the fact that one rarely sees necrosis or spindle-shaped cells in adenomas.

There was no evidence in our study that small-cell cancer could change to another cell type or vice versa.

The evidence that most of the small-cell cancers arise in the larger bronchi seems good; in our series there was only one peripheral tumor in twenty-nine cases in which exploration was done, and in eighty-nine cases in which roentgenograms were available. This is in agreement with the studies of Gebauer,[11] Karsner and Saphir,[18] Koletsky,[19] and others. Few studies have supported the opposite thesis of Tuttle and Womack.[9] This fact of origin in the larger bronchi no doubt helps to explain the high incidence of positive bronchoscopic and cytologic findings in our study.

That these tumors have usually metastasized when one sees them is attested to by the fact that in all cases in our series metastasis either was demonstrable in the surgical specimens or was obvious clinically when the patient was seen. It is felt by many that these tumors metastasize vigorously by lymphatic routes. The evidence of this series seems to favor this view. The data are not complete enough to warrant discussion of metastasis by other routes. Although mediastinal nodes were involved in all the surgical specimens. there was no evidence that the primary lesions extended into the mediastinum more frequently than does bronchogenic carcinoma of other cell types.

In regard to treatment this study revealed discouraging but not hopeless results. In spite of what appeared to be occasional beneficial palliative results of roentgen therapy there were no cures with this method of treatment, nor was there evidence of any particular sensitivity to roentgen therapy.

In our series of fifteen patients who underwent pneumonectomy for bronchogenic carcinoma two patients were living at last report. One patient was living more than five years and one was living two years and nine months after operation. The remaining thirteen patients died at intervals ranging up to twenty-seven months after operation. As our knowledge now stands, small-cell carcinoma carries a very poor outlook. Adams[13] had no survivors in his group of surgically treated patients, and one is hard put to it to find any series where more than one or two five-year survivors exist who had proved small-cell carcinoma. In spite of a poor prognosis we cannot agree with Bariéty and others[20] that patients with this type should not be operated on. Surgical treatment continues to offer the only hope for these patients though small it may be.

For this type of carcinoma our study showed a lower operability rate, but approximately the same resectability rate as for carcinoma without distinction as to cell type. In the past few years it has been the tendency both here and elsewhere to do more palliative resections. This will result, of course, in a higher resectability and operability rate, but it is incumbent upon surgeons to indicate that the operation has been a palliative one, when such it is, as it is only with such honesty that true knowledge will result.

Apparently the rate of operability in regard to such malignant tumors would be lower were it not for the fact that these tumors metastasize first to lymph nodes and later to distant sites. Also, it is believed that probably many patients with inoperable growths of this cell type did not have even bronchoscopy and, hence, were not included in our study. This would make the operability rate found in this study falsely high, and the ratio of bronchogenic carcinoma of the small-cell type to bronchogenic carcinoma of all cell types falsely low.

SUMMARY AND CONCLUSIONS

Ninety cases of small-cell carcinoma of the lung were encountered at the Mayo Clinic in the years 1906 through 1948. These constituted 8.8 per cent of all cases of primary bronchogenic carcinoma in which positive tissue proof of the diagnosis was available. The predominant age of occurrence was in the fifth, sixth, and seventh decades. The predominance of males over females was 29 to 1. Race and occupation were not significant factors.

The average duration of symptoms from onset to diagnosis was 5.9 months. Five patients had symptoms for less than one month. Only three patients had symptoms for as long as two years, and for these the evaluation of true symptoms was difficult. The symptoms were identical with those of bronchogenic carcinoma in general; cough, pain in the chest, dyspnea and episodes of pneumonitis were the most frequent. Physical signs also were those of bronchogenic carcinoma. Dullness over the affected areas, diminished breath sounds, lag in respiration, and râles in the affected areas were the most frequently noted signs.

In all cases in which roentgen examination was made there were positive pulmonary findings with 1 exception in which the interpretation was "negative, possible mediastinal widening." In five cases the interpretation was bronchiectasis, illustrating the danger of accepting the roentgenologic report as the final diagnosis. Further evaluation in all these five cases revealed that the bronchiectasis was secondary to bronchial obstruction by a tumor. There was a slight predominance of lesions in the right side.

Bronchoscopic biopsy gave positive results in 76.5 per cent of cases. In an additional 16 per cent, some evidence of disease was noted but either no lesion was found or biopsy of suspicious areas gave negative results.

Of thirty-one cases in which cytologic examination was done, carcinoma cells were found in twenty-nine, to give a diagnostic accuracy of 93.5 per cent. In more than 70 per cent of the cases in which there was subsequent tissue confirmation it was possible to diagnose the cancer as being of the small-cell type. This indicates that cytologic examination of sputum and bronchial secretions is very helpful when experienced personnel are evaluating the findings.

The pathologic characteristics of small-cell carcinoma are fairly definite and explain in some part the poor prognosis. The only tumors of the lung that offer the pathologist difficulty in differential diagnosis are the atypical adenomas and the true primary lymphosarcoma of the lung. Close study in most cases will reveal the true nature of the tumor.

Of the ninety patients with small-cell bronchogenic carcinoma in this series, twenty-nine underwent exploration, and fifteen of the twenty-nine had pneumonectomy. All the pneumonectomy patients were traced; two were still alive, one five years and four months, and the other two years and nine months, after operation. The last-mentioned patient, however, had a malignant growth removed from the small intestine which probably represented metastasis. All but six of the patients who did not undergo exploration or whose lesion could not be removed surgically were traced; they were all dead. The six untraced patients had definite evidence of hopeless malignancy and must be assumed to be deceased.

Roentgen therapy was given to seventeen patients either at the clinic or in the patient's home community. While several seemed to get good palliative results, there were no survivors in this group.

This study indicates, in our opinion, that small-cell bronchogenic carcinoma is a definite entity and should not be included in the general group of anaplastic or undifferentiated cancer, even though admittedly its behavior is of such a nature. This opinion is based on the fact that there exist (1) a definite, easily recognizable, microscopic picture, (2) a definite high incidence of cytologically positive results on sputum examination which makes the condition easily recognized and diagnosed, and (3) a characteristic clinical behavior.

Finally the results of this study bear out the succinct statement of Dr. Edward Churchill[21]: "The prognosis following surgical extirpation has not been fully correlated with the predominant cytologic characteristics of cancers of the lung. The evidence that exists, however, confirms the reasonable hypothesis that growths with highly undifferentiated cells are rapidly invasive and tend to metastasize early. The same may be said for the oat cell type. Significant results achieved by the surgical extirpation of cancers of these morphologic types are rare. A biopsy that demonstrates highly undifferentiated or oat cell cancer is presumptive evidence that complete surgical extirpation is a remote possibility."

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BRONCHOGENIC SQUAMOUS-CELL CARCINOMA*

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THE microscopic classification of primary bronchogenic carcinoma now in use at the Mayo Clinic consists of four subdivisions: squamous-cell carcinoma, adenocarcinoma, large-cell carcinoma and small-cell carcinoma. Definite microscopic criteria have been set up for each of these groups. The microscopic criterion required in this study to classify any primary bronchogenic tumor as squamous-cell carcinoma was (1) the presence of intracellular bridges, or (2) definite epithelial pearl formation, or (3) definite keratinization or squamatization, or (4) definite polarization of cells (a whorling tendency) without true pearl formation if accompanied either by minimal squamatization or keratinization or by the typical growth pattern of squamous-cell carcinoma. If any of the first three criteria was found in a single area of any section the tumor was classified as squamous-cell carcinoma. In order to classify a tumor on the basis of only the last criterion, however, more than one area showing the specified characteristics was required.

The characteristic growth pattern of this tumor consists of anastomosing and branching columns of epithelial cells encroaching on the stroma (Fig. 1*a* and *b*.) There is a tendency for the epithelial cells to line up along the edges of these columns, as if in an attempt to form a growing edge of skin. Many squamous-cell carcinomas, however, do not exhibit this growth pattern, and conversely, many large-cell carcinomas do.

Many specimens previously labeled "squamous-cell carcinoma" did not meet the foregoing requirements for this diagnosis. The majority of these were reclassified as large-cell carcinomas, and it was with this group that the major difficulty in classification occurred.

MATERIAL AND METHODS

The records of all cases in the files of the Mayo Clinic through 1949 in which a diagnosis of a tumor of the lung or bronchus had been made and in which positive microscopic proof obtained by bronchoscopy or thoracotomy was available were reviewed. This group involved a study of about 1,400 cases. Of this group, 373 cases met the criteria for primary squamous-cell carcinoma of the bronchus. This study is an analysis of these 373 proved cases.

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Pulmonary resection was performed in 121 cases: in 42 cases of this group resection was performed more than five years prior to the time of this inquiry, and in 89 cases it was done more than two years prior to inquiry. More than 90 per cent of the patients who survived operation were traced.

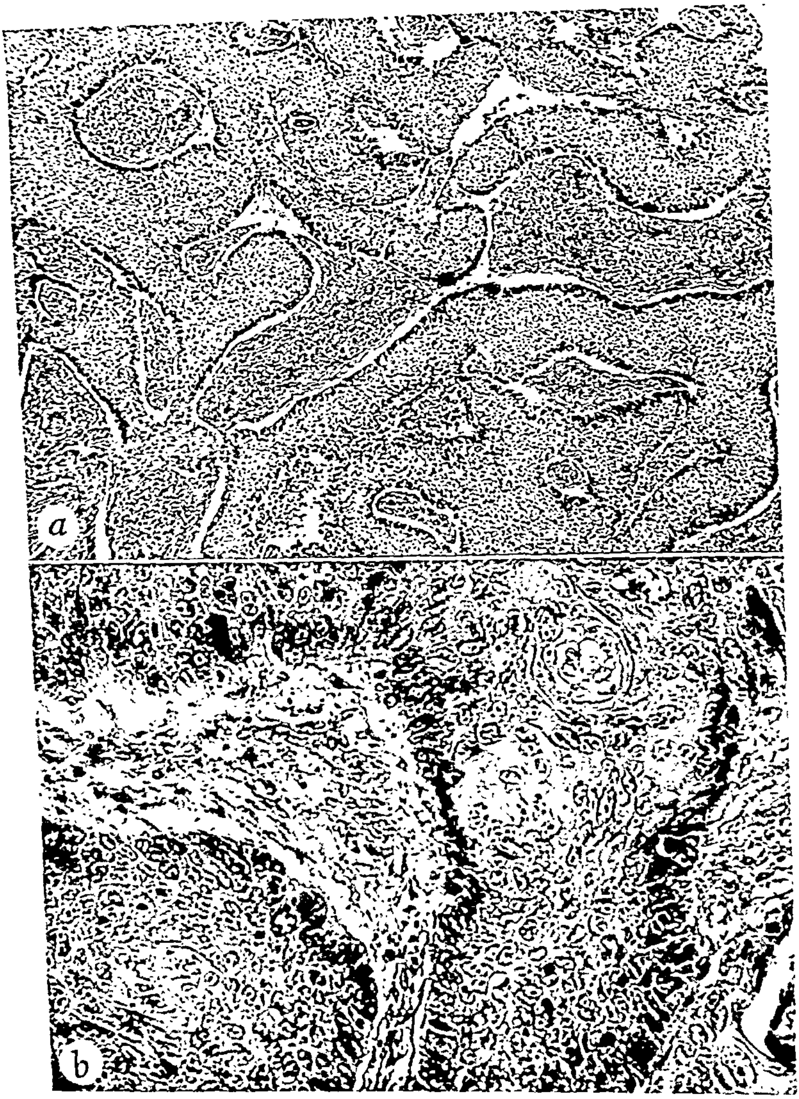

Fig. 1.—Typical growth pattern. Sections stained with hematoxylin and eosin *a*. (×45) *b*. (×200.)

Up through December, 1948, there were 849 proved cases of bronchogenic carcinoma, of which 321 were of the squamous-cell variety, giving an incidence of 37.8 per cent, as compared with 8.8 per cent for small-cell carcinoma, 13.2 per cent for adenocarcinoma, and 40.2 per cent for large-cell carcinoma, for the same period.

PATHOLOGIC CHARACTERISTICS

The 373 squamous-cell carcinomas were graded according to the highest grade of malignancy seen in any section. With this method, 99.5 per cent of the tumors were classified as grade 3 or grade 4 carcinomas. It is not surprising, therefore, that only 13.8 per cent showed true epithelial pearl formation. This hallmark of squamous-cell epithelioma is more characteristically seen in lower grade tumors.

Microscopically the tumor spread mainly in the extracartilaginous portion of the bronchial wall, at times pushing up through the cartilage plates to form an intrabronchial nodule proximal to the main lesion. This is illustrated in Fig. 2, a section separated by 4 mm. of normal mucosa from the main intrabronchial lesion. In situ squamous-cell carcinoma of the bronchial mucosa was also noted in several instances proximal to the gross lesion in the bronchus.

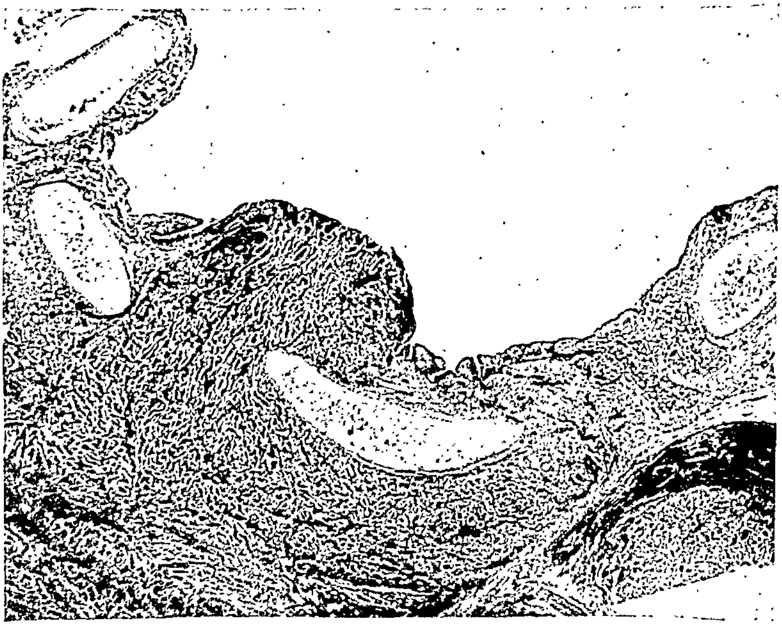

Fig. 2.—View of a "skip" area in an involved bronchus (hematoxylin and eosin ×7.5).

This is not as much a hazard, from the surgeon's viewpoint, as might be anticipated, because in the majority of cases there was obvious gross carcinoma surrounding the bronchus deep to the normal-appearing bronchial mucosa.

Grossly the majority of tumors presented a similar pattern. There was an ulcerated or nodular involvement of a central bronchus with an elliptical mass of carcinoma proximal to and often surrounding the involved bronchus. Lymph nodes were frequently invaded by this mass, but much less frequently were lymph nodes proximal to the gross lesion involved with tumor. Of the 121 cases in which resection was performed, lymph nodes were involved in 43.7 per cent. The average size of the gross tumor was between 2 by 2 by 2 cm. and 5 by 5 by

5 cm., but in some cases the masses were considerably larger, and characteristically more peripherally located, with the involved bronchus seeming to "run into" the mass. It was this type of tumor which sometimes showed a necrotic central abscess. The incidence of abscess formation, however, was only 13 per cent in the group in which resection was performed, and about half of these were in nonmalignant tissue distal to an obstructed bronchus.

The primary growth was located in the right lung in 57.9 per cent of the 121 cases in which resection was performed. The two most frequently involved lobes were the right lower lobe with thirty-five cases and the left upper lobe with thirty cases. In only four cases or 3.3 per cent was the tumor truly peripheral in location in that it lay more than 4 cm. from the main bronchus or its continuation as the main lower lobe bronchus. Sixty-five and three-tenths per cent of the tumors were in the primary or secondary bronchi, and two-thirds of these were in the main bronchi or the main lower lobe bronchi (Fig. 3).

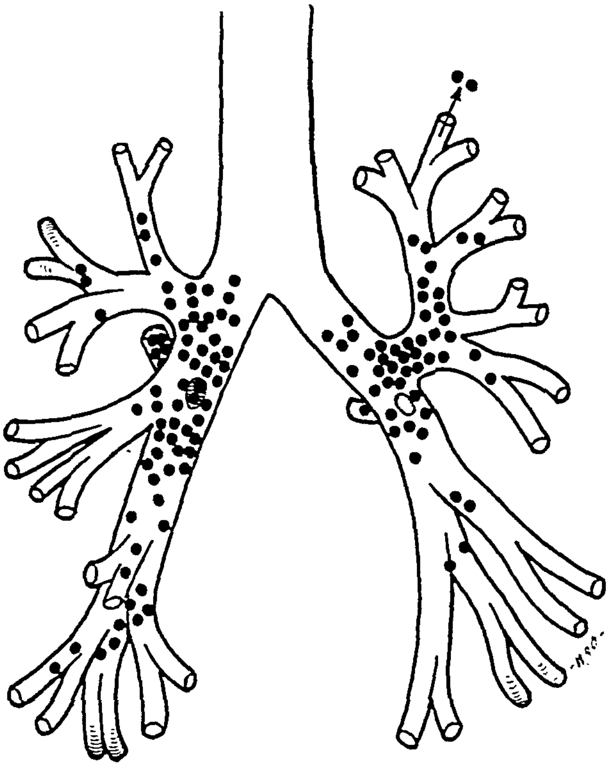

Fig. 3.—Site of the primary growth in 121 cases of squamous-cell bronchogenic carcinoma in which resection was performed.

DIAGNOSIS

Bronchoscopy was performed in more than 99 per cent of the 373 cases, but gave positive results in only 64.5 per cent of the 121 cases in which resection was performed. More than 50 per cent of all tumors located in the right upper lobe, right middle lobe, or left upper lobe gave negative results on bronchoscopic biopsy, and 44 per cent of those located in the secondary branches of the right lower lobe also gave negative results on bronchoscopic biopsy. In the series in which resection was done, bronchoscopic biopsy gave positive results in 71 per cent of the tumors in the right lung, but in only 53 per cent of those in the left side.

Roentgenologic examination of the chest proved to be the most constant means of determining an abnormal condition in this series. The results of more than 99 per cent of such examinations for the entire group were reported as abnormal. Unfortunately, these findings were rarely diagnostic. The most common findings were obstructive pneumonitis in 42.6 per cent, hilar mass in 28.8 per cent, infiltration of a lobe or part of a lobe in 15.5 per cent, and thickened pleura or bronchiectasis in 10.9 per cent.

In addition to bronchoscopy and the roentgen-ray examination of the chest, a third and more recent addition to the available diagnostic aids in bronchogenic carcinoma is the examination of the sputum or bronchial secretions for malignant cells. Cytologic studies were made in 116 cases in this series including 95 per cent of all the cases encountered from Jan. 1, 1948, through December, 1949. In 72.4 per cent of these 116 cases cytologic studies gave positive results. One hundred patients had cytologic examination of the sputum, with positive results in 63 per cent, and seventy-six patients had cytologic examination of bronchial washings with positive results in 59 per cent.

The clinical findings in this series of cases differed very little from those of most other reported series of bronchogenic carcinoma except that there were fewer patients under 40 years of age in this series. The age range was from 37 to 71 years. The age distribution in this series is shown in Table I.

There were only 13 females (3.5 per cent) in the 373 cases. In the entire group of proved cases of primary bronchogenic carcinoma 10.3 per cent were females.

The duration of symptoms before the definitive diagnosis was established varied greatly but averaged about eight months. The duration in the group with resection was not markedly different from that in the group without resection.

Cough, sputum, hemoptysis, loss of more than 10 pounds, pain in the chest, dyspnea, and fever each occurred in more than 40 per cent of patients in both the group with resection and the group without resection. The presence of pain as a chief complaint and the presence of hoarseness or osteo-arthropathy were each associated with a high incidence of inoperability.

TABLE I. AGE DISTRIBUTION IN 373 PROVED CASES OF PRIMARY SQUAMOUS-CELL BRONCHOGENIC CARCINOMA

AGE, YEARS	CASES	PER CENT OF TOTAL CASES
30-39	6	1.6
40-49	72	19.3
50-59	175	46.9
60-69	117	31.4
70-79	3	0.8
Total	373	100.0

PROGNOSIS WITHOUT PULMONARY RESECTION

The survival rate* for patients without pulmonary resection is shown in Table II for 204 patients who were adequately followed for the indicated period of time. One hundred ninety-two patients were followed two years, and only seven, or 3.6 per cent, survived for two years after the definitive diagnosis was established. There were no three-year survivals.

SURGICAL TREATMENT AND RESULTS

Exploratory thoracotomy was performed on 177 patients. The operation was performed on or after Jan. 1, 1940, in 173 cases, to give an operability rate of 57.7 per cent in the ten-year period from 1940 through 1949. From Jan. 1, 1945, through Dec. 31, 1949, 124 of 175 patients with pathologically proved squamous-cell carcinoma had exploratory thoracotomy. This represents an operability rate of 71 per cent. The significance of these figures is open to some question, however, because cases of bronchogenic carcinoma not verified by biopsy were not included in this series. Perhaps of more importance is the resectability rate in those cases in which exploratory thoracotomy was done. For the ten-year period from 1940 through 1949 pulmonary resection was performed in 120 of 173 thoracotomies, giving a resectability rate of 69.4 per cent. From 1945 through 1949 the resectability rate was 71 per cent.

Pulmonary resection was performed in 121 cases. In forty-two cases the operation was performed more than five years prior to inquiry, and in eighty-nine cases more than two years prior to inquiry. More than ninety per cent of the patients who survived operation were adequately followed. Table III gives the two-year and five-year survival rates for the group with pulmonary resection.

TABLE II. PROGNOSIS FOR 204 PATIENTS WITHOUT PULMONARY RESECTION

PERIOD (MONTHS)	NO OPERATION				EXPLORATORY THORACOTOMY ONLY			
	PATIENTS		LIVED BEYOND INDICATED PERIOD		PATIENTS		LIVED BEYOND INDICATED PERIOD	
	TOTAL	TRACED	NUMBER	PER CENT*	TOTAL	TRACED	NUMBER	PER CENT*
6	195	161	83	51.6	53	43	29	67.4
12	195	159	28	17.6	53	40	11	27.5
18	190	145	13	9.0	49	37	4	10.8
24	187	156	5	3.2	42	36	2	5.6
36	179	151	0	0.0	38	32	0	0.0

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included in the twelve-month group are only those patients operated on twelve months or more prior to the time of inquiry, that is, operated on in 1949 or earlier; included in the eighteen-month group are those patients operated on June 30, 1949, or earlier, and so forth. Hospital mortality is excluded in the calculation of survival rates.

There were seventeen hospital deaths in the 121 cases in which resection was done, giving a hospital mortality rate of 14 per cent. Seventy-nine resections were performed from Jan. 1, 1946, through December, 1949, with only five hospital deaths, giving a hospital mortality rate of 6.3 per cent.

Therefore, given 100 patients with proved primary squamous-cell bronchogenic carcinoma, it can be said on the basis of this study that 71 will be operable, 50 will have resectable lesions, 47 will survive operation, and 24 will be alive in five years.

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*The calculations of the survival rates were made by the direct method as described by Berkson and Gage (Proc. Staff Meet., Mayo Clin. 25: 270-286, 1950).

TABLE III. SURVIVAL RATES FOLLOWING PULMONARY RESECTION

PERIOD,	PATIENTS		LIVED BEYOND INDICATED PERIOD	
YEARS	TOTAL	TRACED	NUMBER	PER CENT*
2	75	68	38	55.9
5	30	27	14	51.9

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included in the two-year group are only those patients operated on two or more years prior to the time of inquiry, that is, operated on in 1948 or earlier; included in the five-year group are those patients operated on in 1945 or earlier. Hospital mortality is excluded in the calculation of survival rates.

FACTORS AFFECTING PROGNOSIS

Because of the unexpectedly high survival rate following pulmonary resection, an attempt was made to analyze various factors to determine if they were of prognostic importance. Neither the age of the patient, the duration of symptoms, nor any of the clinical findings was found to affect the prognosis significantly. Similarly, the microscopic grade of the tumor did not affect the prognosis. Since more than 99 per cent of the entire group of tumors had areas of grade 3 or grade 4 carcinoma present, it is doubtful that grading of these tumors is of significance.

Patients with tumors in the right lung had a slightly higher survival rate than those with left-sided lesions. The two-year survival rates for these groups are shown in Table IV.

TABLE IV. THE RELATIONSHIP OF RIGHT-SIDED AND LEFT-SIDED LESIONS TO PROGNOSIS

LOCATION OF	PATIENTS		LIVED 2 OR MORE YEARS AFTER OPERATION	
LESION	TOTAL	TRACED	NUMBER	PER CENT*
Right	44	39	25	64.1
Left	31	29	13	44.8
Total	75	68	38	55.9

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included are patients operated on two or more years prior to the time of inquiry, that is, operated on in 1948 or earlier. Hospital mortality is excluded in the calculation of survival rates.

TABLE V. THE RELATIONSHIP OF INVOLVEMENT OF THE REGIONAL LYMPH NODES TO PROGNOSIS

	TWO-YEAR FOLLOW-UP				FIVE-YEAR FOLLOW-UP			
	PATIENTS		LIVED TWO OR MORE YEARS AFTER OPERATION		PATIENTS		LIVED FIVE OR MORE YEARS AFTER OPERATION	
LYMPH NODE INVOLVEMENT	TOTAL	TRACED	NUMBER	PER CENT*	TOTAL	TRACED	NUMBER	PER CENT*
Positive	28	24	10	41.7	10	8	2	25.0
Negative	47	44	28	63.6	20	19	12	63.2
Total	75	68	38	55.9	30	27	14	51.9

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included in the two-year group are only those patients operated on two or more years prior to the time of inquiry, that is, operated on in 1948 or earlier; included in the five-year group are those patients operated on in 1945 or earlier. Hospital mortality is excluded in the calculation of survival rates.

Regional lymph nodes were involved with carcinoma in 43.7 per cent of the cases in which resection was performed. A sharp fall in the survival rate of those patients with positive findings in the regional lymph nodes was demonstrated only in the five-year follow-up studies, and then in an inadequate number of cases to be significant. Table V shows the two-year and five-year survival rates for those patients with and without involvement of the regional lymph nodes.

Another factor which seemed to affect the prognosis adversely was the presence of an abscess in the resected specimen. This occurred in 12.4 per cent, and of the eleven patients followed two or more years, only four, or 36.4 per cent, lived two years.

The most significant prognostic finding, however, was the size of the gross lesion. The two-year survival rate for patients with gross lesions smaller than 2 by 2 by 2 cm. was 78.9 per cent, while only 23.1 per cent of patients with lesions larger than 5 by 5 by 5 cm. survived two years (Table VI).

It was then noted that many patients with large gross lesions had a hilar mass reported on their chest roentgenogram, while many patients with small lesions had obstructive pneumonitis but no hilar mass on their chest roentgenogram. Table VII shows the two-year survival rates for patients with various roentgenologic findings in the chest.

TABLE VI. RELATIONSHIP OF THE SIZE OF THE GROSS LESION TO PROGNOSIS

SIZE OF GROSS LESION	PATIENTS		LIVED TWO OR MORE YEARS AFTER OPERATION	
	TOTAL	TRACED	NUMBER	PER CENT*
Smaller than 2 by 2 by 2 cm.	23	19	15	78.9
From 2 by 2 by 2 to 5 by 5 by 5 cm.	39	36	20	55.6
Larger than 5 by 5 by 5 cm.	13	13	3	23.1
Total	75	68	38	55.9

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included are patients operated on two or more years prior to the time of inquiry, that is, operated on in 1948 or earlier. Hospital mortality is excluded in the calculation of survival rates.

These findings strongly suggest that patients with small partially obstructing tumors, shown in the roentgenogram of the chest by obstructive pneumonitis, have a two-year survival rate of approximately 80 per cent. All these patients should therefore have repeated sputum studies and bronchoscopy in an effort to determine if the tumor is of the squamous-cell variety. If so, a very favorable prognosis may be given.

TABLE VII. RELATIONSHIP OF THE ROENTGENOLOGIC FINDINGS IN THE CHEST TO PROGNOSIS

ROENTGENOLOGIC FINDINGS	PATIENTS		LIVED 2 OR MORE YEARS AFTER OPERATION	
	TOTAL	TRACED	NUMBER	PER CENT*
Hilar mass	23	19	9	47.4
Atelectasis without hilar mass	17	16	13	81.3
All others	35	33	16	48.5
Total	75	68	38	55.9

*Based on traced patients. Inquiry as of Jan. 1, 1951. Included are patients operated on two or more years prior to the time of inquiry, that is, operated on in 1948 or earlier. Hospital mortality is excluded in the calculation of survival rates.

SUMMARY

On the basis of definite criteria set up for the diagnosis of primary squamous-cell bronchogenic carcinoma, records of 373 microscopically proved cases were found in the surgical files of the Mayo Clinic through December, 1949. Thirty seven and eight-tenths per cent of all primary bronchogenic carcinomas in this series were squamous-cell in type. Squamous-cell bronchogenic carcinoma rarely occurred under the age of 40. Only 3.5 per cent were females. Bronchoscopy gave positive results in 64.5 per cent of 121 cases in which resection was done. Cytologic examinations gave positive results in 72.4 per cent of 116 cases.

The two-year survival rate in the absence of resection was 3.6 per cent. No patient survived three years without pulmonary resection.

In the five-year period from 1945 through 1949, 71 per cent of 175 patients with pathologically proved lesions had exploratory thoracotomy, and 71 per cent of the 124 thoracotomy patients had pulmonary resection. Both the five-year survival rate and the two-year survival rate for those patients who survived pulmonary resection were more than 50 per cent. Left-sided lesions, lung abscess, and involvement of the regional lymph nodes all affected the prognosis adversely, but the finding of greatest prognostic importance was the gross size of the lesion.

The hospital mortality rate was only 6.3 per cent for seventy-nine pulmonary resections performed from Jan. 1, 1946, through December, 1949.

BRONCHOGENIC ADENOCARCINOMA

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THE differentiation of adenocarcinoma from the other types of bronchogenic carcinoma is based on the microscopic evidence of glandular formation or function. The criterion used is the finding of definite alveolar, acinar, or papillary structures or the formation of extracellular or intracellular mucus, with columnar or cuboidal configuration of the cells (Fig. 1). Of course, all general criteria of malignancy must be met. It should be emphasized that the adenocarcinoma pattern should be predominant or, at the very least, present in a significant number and area of the microscopic sections.

Three specific pitfalls to the correct diagnosis should be mentioned. First, bronchial adenomas should be excluded because of their different origin and behavior. Second, alveolar-cell carcinomas, although probably a definite entity, should be excluded because they arise from the alveolar wall and are therefore not strictly bronchogenic. Third, the bronchial glands in the vicinity of any neoplasm have a tendency to become hyperplastic and disorganized, and, unless care is taken, their appearance may lead one to a false diagnosis of adenocarcinoma.

THE PRESENT STUDY

To the best of our knowledge, all cases in which there was a primary carcinoma elsewhere in the body have been excluded, whether or not we felt that the bronchial lesion was primary. Therefore we believe that the study concerns only the cases of primary adenocarcinoma of the bronchial epithelium. This study is based on 132 cases of adenocarcinoma from the surgical pathology laboratories of the Mayo Clinic through 1949. In forty cases the specimens of tissue were obtained at resection, and in ninety-two cases they were obtained at biopsy, sixty-seven of which represented bronchoscopic biopsy only.

There were 849 proved cases of primary bronchogenic carcinoma through 1948, 112 of which were adenocarcinoma, giving a ratio of 13.2 per cent. This contrasts markedly with other series in the literature in which a higher ratio is shown, but it must be remembered that bronchial adenomas, a large-cell group, and any lesions that might have been metastatic were eliminated.

The youngest patient in this group was 31 years of age, and the oldest 70 years. The average age was 53.3 years. Table I shows the age distribution by decades. It can be seen that more than 90 per cent of the patients were in the age group 40 to 69 years, and nearly half were in the age group 50 to 59 years. There were twenty-six women, representing 19.7 per cent of the total.

In the forty cases in which specimens obtained at resection were available, the point of origin of the tumor was determined as closely as possible. It was decided to regard those lesions in the primary and secondary bronchi as central, and those in the tertiary bronchi or beyond as peripheral (Fig. 2). With this scheme of classifying the tumors, twenty-six of the forty cases in which resection

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TABLE I. AGE DISTRIBUTION IN 132 CASES OF BRONCHOGENIC ADENOCARCINOMA

AGE (YEARS)	CASES	PER CENT
30-39	12	9.1
40-49	28	21.2
50-59	56	42.4
60-69	35	26.5
70	1	0.8
TOTAL	132	100.0
MEAN AGE	53.3 YEARS	

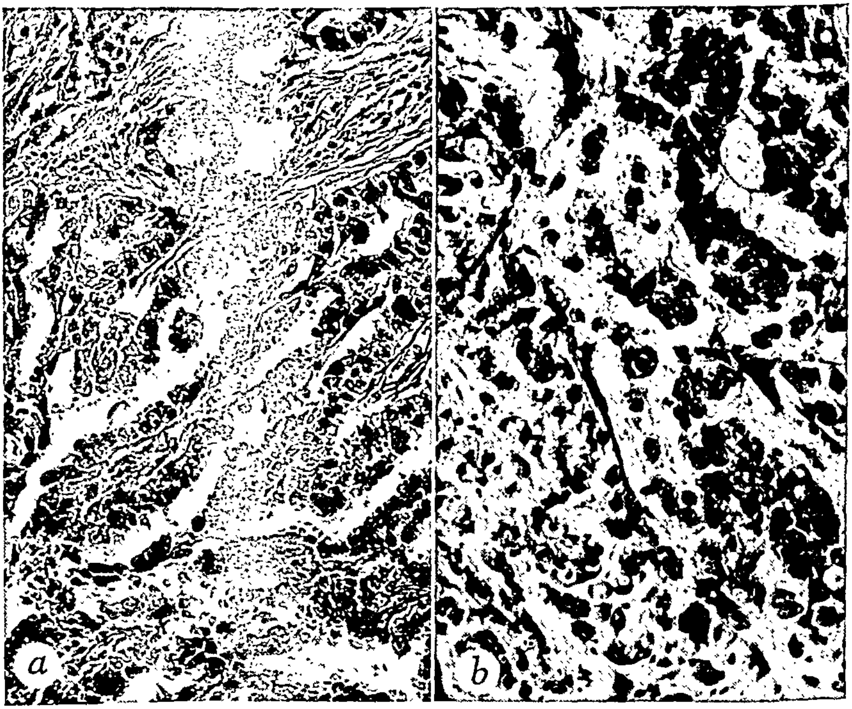

Fig. 1.—*a*, Papillary type of adenocarcinoma (hematoxylin and eosin ×150). *b*, Mucus-producing type of adenocarcinoma with both extracellular mucus and signet-ring cells (hematoxylin and eosin ×160).

was performed (65 per cent) were found to represent peripheral lesions, and fourteen cases (35 per cent) represented central lesions (Fig. 3).

Thirty-six of the patients underwent bronchoscopy prior to operation; in nine of these bronchoscopic biopsy gave positive results. All those with positive results on biopsy had lesions that were judged as being centrally located when the gross surgical specimen was examined. While these nine patients with positive results on biopsy represented only 25 per cent of the total patients who underwent bronchoscopy, they nevertheless represented 64.3 per cent of the cases in which the lesions were centrally situated.

The symptoms were in generally those of any type of bronchogenic carcinoma. The five most prominent symptoms were cough, pain, loss of weight, hemoptysis, and dyspnea in the following incidence: cough, 83.8 per cent; pain or aching in the chest, 49.6 per cent; loss of weight, 48.7 per cent; blood-streaked sputum or hemorrhage, 47.0 per cent; and dyspnea, 24.1 per cent. The average duration of symptoms from onset to histologic diagnosis was 7.9 months for the cases in which biopsy only was performed, either bronchoscopically or surgically, and 6.8 months for the cases in which resection was done. Details are given in Table II.

Cytologic examination of the sputum and bronchial secretions for malignant cells was done in fifty-one cases, and such cells were found in thirty-two cases (62.7 per cent). Of the nineteen cases in which negative results were obtained, in twelve the examinations were judged inadequate, that is, less than three specimens for cytologic examination were received in the laboratory. With these cases excluded, cytologic examination gave positive results in 82.1 per cent of the adequate examinations.

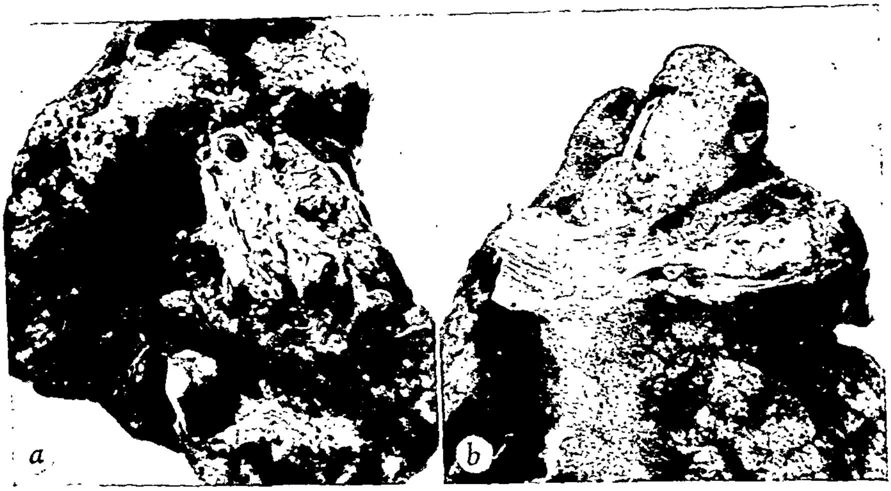

Fig. 2.—*a*, Typical central bronchogenic adenocarcinoma in the bronchi of the main and lower lobes of the right lung. *b*, Typical peripheral bronchogenic adenocarcinoma in the apex of the left lung.

From 1940 through 1944 there were forty-four cases; in thirteen of these (29.5 per cent) exploration was carried out, and in five cases (11.4 per cent) resection was done. Thus resection was done in 38.5 per cent of the cases in which exploration was carried out. From 1945 through 1949 there were sixty-seven cases, with forty-six explorations and thirty-four resections. This gives an exploration rate of 68.7 per cent, and a resection rate of 50.7 per cent. In 73.9 per cent of the cases in which exploration was done, resection was carried out. Apparently the higher resection rate of the latter group is due partially to a higher rate of exploration and partially to an increase in the cases selected for resection at exploration.

In three of the cases, reports of roentgenologic examination were not available. In all the remainder, abnormalities were found in the roentgenograms of the chest, but it was felt that in 12 per cent of these cases bronchogenic carcinoma was not suspected. In the cases in which it was judged that carcinoma was unsuspected on roentgenologic examination, such diagnoses as "tuberculosis," "empyema with tuberculosis," and "bronchiectasis" were the most common. In the remainder of the cases, or 88 per cent, it was felt that bronchogenic carcinoma was either suspected or should have been. Such diagnoses as "mass in the right midlung field,'' ''bronchial obstruction with atelectasis,'' or ''atelectasis'' alone, as well as ''bronchogenic carcinoma'' or ''rule out malignancy'' were accepted as evidence that primary bronchogenic carcinoma was suspected.

On attempting to grade the lesions according to the method of Broders, we were immediately faced with the problem that the individual cases not only varied as to cell type throughout the lesion, but that they varied likewise as to grade. It was felt, however, that the grade should be called according to the highest grade found in the lesion, since presumably that portion of the tumor would be the most malignant and the most likely to metastasize. It was found that there was rarely more than one grade difference in an individual tumor;

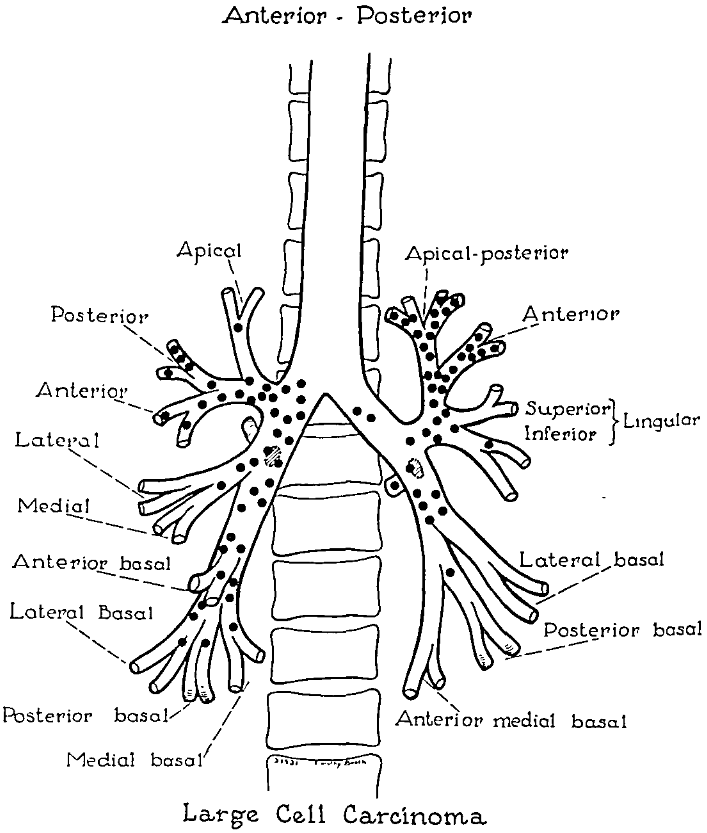

Fig. 3.—Position of the tumor in forty cases of bronchogenic adenocarcinoma in which resection was performed (anteroposterior view). Each dot represents an individual case.

TABLE II. DURATION OF SYMPTOMS IN 132 CASES OF BRONCHOGENIC ADENOCARCINOMA

DURATION OF SYMPTOMS, MONTHS	SOURCE OF SPECIMEN	
	BIOPSY, PER CENT OF 92 CASES	RESECTION, PER CENT OF 40 CASES
ASYMPTOMATIC	2.2	20.0
1-3	17.4	22.5
4-6	26.1	22.5
7-9	21.7	10.0
10-12	20.7	5.0
13-24	12.0	17.5
25-36	0	2.5
MEAN	7.9 MONTHS	6.8 MONTHS

that is, if we found a tumor which we graded 3, there might be and usually was a significant portion of the tumor which could be graded 2, but rarely any portion of the tumor which could be graded 1. Using this system we found that 117 of the 132 lesions, or 88.6 per cent, were grade 3 and grade 4, while fourteen lesions, or 10.6 per cent of the total, were grade 2, and one lesion was grade 1. Thus most of the patients had high grade lesions.

Pneumonectomy or lobectomy for carcinoma of the bronchus was performed on forty patients of this series, with six hospital deaths (15 per cent). These operations are rather recent procedures in surgery, and this fact was evident when we tried to get an idea about prognosis in this group. Only sixteen patients in the series were operated on prior to 1947, and nearly half of the patients were operated on in 1948 or 1949. For this reason we felt that prognosis* could be logically calculated only on a short-term basis (Table III). More than one-half of the patients who were operated on are alive at the end of one year, and a third will be alive two years after operation. This indicates that the prognosis in this type of bronchogenic carcinoma is poor, but it will take the test of time to prove whether or not the prognosis is as bad as is indicated by this series. A breakdown of the seven patients who survived two or more years following operation revealed that three patients died during the third year following operation, and the remainder were still alive: one was alive during the third year following operation, two were alive during the fifth year, and one was alive more than ten years following operation. This at least indicates that though the prognosis may be bad it is not hopeless, and some patients may survive for relatively long periods after operation.

TABLE III. SURVIVAL RATES FOR PATIENTS WITH BRONCHOGENIC ADENOCARCINOMA FOLLOWING PNEUMONECTOMY OR LOBECTOMY

PERIOD, YEARS	PATIENTS		LIVED BEYOND INDICATED PERIOD	
	TOTAL	TRACED	NUMBER	PER CENT*
1	34	28	16	57.1
2	23	21	7	33.3

*Based on traced patients, inquiry as of Jan. 1, 1951. Included in the one-year group are only those patients operated on one or more years prior to the time of inquiry, that is, operated on in 1949 or earlier; the two-year group includes only those patients operated on in 1948 or earlier. Hospital mortality is excluded in the calculation of survived rates.

SUMMARY

The criteria for diagnosis of bronchogenic adenocarcinoma have been presented, and certain aspects of diagnosis reviewed. It was found that 13.2 per cent of all primary carcinomas in this series were adenocarcinomas, and that about one-fifth of the patients were females. The majority of tumors were of peripheral origin which was reflected in the low rate of positive results on biopsy. Cytologic examination gave positive results in about 80 per cent of cases. Apparently symptoms, age distribution, and roentgenologic findings were similar to those of other types of bronchogenic carcinoma. The prognosis is apparently poor, since two years after operation two-thirds of the patients who survive resection die.

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*The calculations of the survival rates were made by the direct method as described by Berkson and Gage (Proc. Staff Meet., Mayo Clin. 25: 270-286, 1950).

BRONCHOGENIC LARGE-CELL CARCINOMA

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THE microscopic diagnosis of the large-cell type of bronchogenic carcinoma is primarily a diagnosis of exclusion. In examination of the tissue in any large group of cases of bronchogenic carcinoma, it is possible to separate a group of typical squamous-cell carcinomas and a group of small-cell carcinomas according to the microscopic criteria given elsewhere in this series of papers. We chose to adhere strictly to the criteria given for small-cell carcinomas, and to put in the squamous-cell group every case in which there was definite evidence of squamous-cell criteria as given elsewhere. Separation of these two groups leaves one with a group of adenocarcinomas and a group in which a specific type of carcinoma cannot be diagnosed with any certainty. For the latter cases a diagnosis of high-grade carcinoma, type indeterminate, would seem to be the most valid one. It is these cases that we have chosen to call "large-cell carcinomas," both because by this means it is possible to bring them together for easy reference, and because in most of the cases the lesions are characterized by being composed of relatively large cells. Unfortunately, however, the border of differentiation between adenocarcinoma and large-cell carcinoma cannot be so easily defined as that between large-cell carcinoma and squamous-cell carcinoma, or between large-cell carcinoma and small-cell carcinoma. We have chosen, therefore, to be as strict as possible in defining adenocarcinomas by saying that the adenocarcinoma pattern must be present in a majority of the microscopic sections and in a significantly large area as stated in another paper of the series.

The cellular pattern of the large-cell group is bizarre with marked variation in the staining qualities and size of the individual cells. The nuclei tend to be large and rather dark with prominent nucleoli, there may be many mitotic figures, and there may be giant-cell forms. The cells may be angular, oval, or spindle-shaped, and they tend to be closely packed in a medullary or pseudoalveolar arrangement. Nearly always there are areas of necrosis which may show fragmented bits of cells intermingled with leucocytes, which may be hemorrhagic, which may be fibrotic, or which may show any combination of these. There also is usually evidence of a highly malignant potential, both as to local destruction of tissue and as to invasion of lymphatics and blood vessels (Fig. 1).

THE PRESENT STUDY

In this study all cases in which there was a primary carcinoma elsewhere in the body were eliminated to the best of our knowledge, whether or not we

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felt that the bronchial lesion was primary. The study is based on 384 cases of large-cell carcinoma encountered in the surgical pathology laboratories of the Mayo Clinic through 1949. These were made up of 87 cases in which the specimens were obtained at resection and 297 cases in which they were obtained for biopsy, 255 representing bronchoscopic biopsy only.

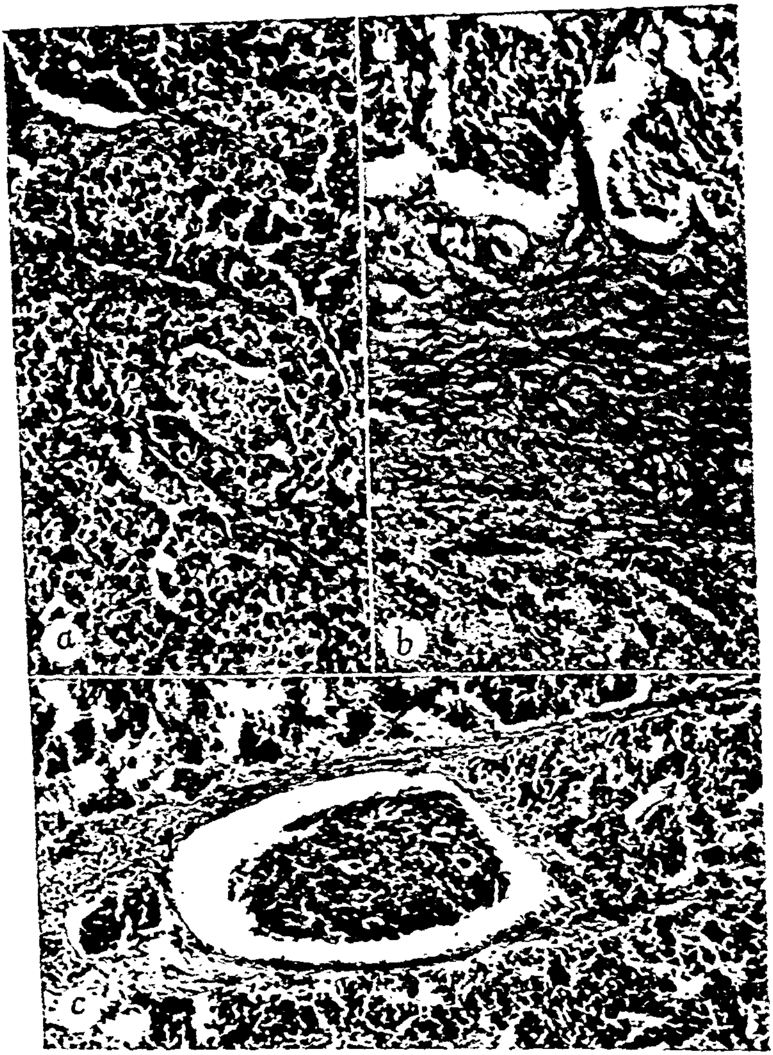

Fig. 1.—*a*, A typical large-cell carcinoma with punched-out areas of necrosis, making the section resemble an adenocarcinoma (hematoxylin and eosin ×110). *b*. A large-cell carcinoma showing necrosis and fibrosis. This was taken from a large area of necrosis, and shows the border of the malignant cells (hematoxylin and eosin ×110). *c*. A large-cell carcinoma showing a plug of carcinoma cells in a lymphatic channel (hematoxylin and eosin ×110).

Through 1948, 849 proved cases of bronchogenic carcinoma had been found at the clinic, of which 341, or 40.2 per cent, were of the large-cell type. Large-cell carcinoma made up the largest group, squamous-cell carcinoma constituting 37.8 per cent, adenocarcinoma 13.2 per cent, and small-cell carcinoma 8.8 per cent of the total.

In the series of 384 cases the youngest patient was 22 years old, the oldest 80 years of age. The average age was 55 years. The age distribution by decades is given in Table I. It can be seen that more than 90 per cent of the patients were in the age group 40 to 69 years, and nearly half were in the group 50 to 59 years. This is about the same ratio as that for the other types of bronchogenic carcinoma, nearly all the patients being over 40 years of age.

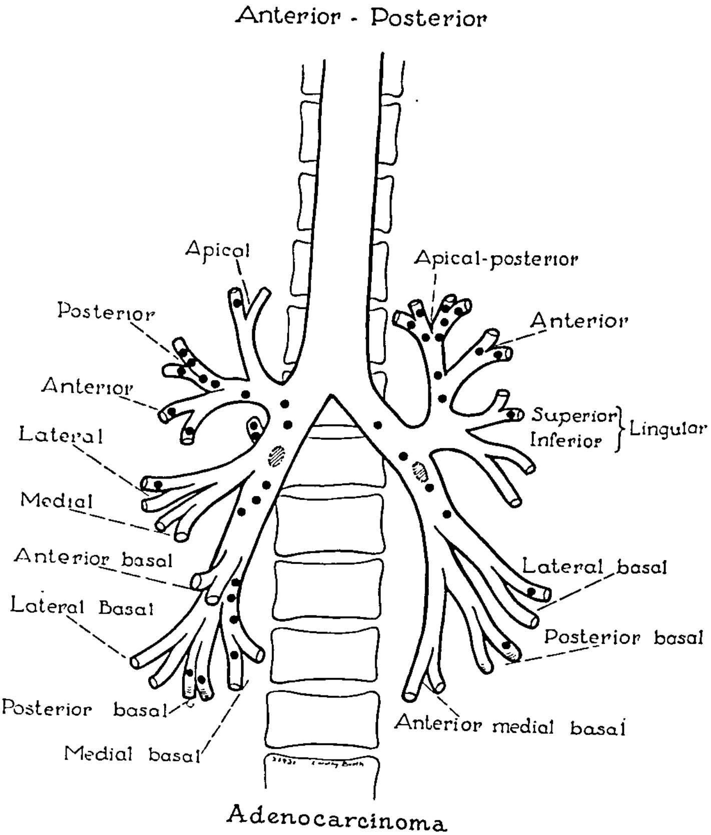

Fig. 2.—Location of the lesion in eighty-five of the eighty-seven cases of resection in which location could be determined (anteroposterior view).

There were fifty-six females in the group, representing 14.6 per cent of the total. This is close to the percentage of 19.7 for females with adenocarcinoma, and it contrasts markedly with the percentage of 3.5 for females with squamous-cell carcinoma and 3.3 for females with small-cell carcinoma.

The eighty-seven surgical specimens were examined grossly. Lesions in the primary or secondary bronchi were classified as central, and those in the tertiary bronchi or beyond as peripheral. It was found that fifty lesions, or 57.5 per cent, were central and that thirty-seven, or 42.5 per cent, were peripheral, giving a ratio of 1.4 to 1. The ratio of central to peripheral lesions for each of the other three types of bronchogenic carcinoma was as follows: small-cell carcinoma 27.7 to 1; squamous-cell carcinoma, 1.9 to 1; and adenocarcinoma, 0.54 to 1. In most of the cases the lesion was in the right main, upper, and lower lobe bronchi and in the left upper lobe (Fig. 2). The left lower lobe was apparently rarely involved. This same phenomenon was observed in the adenocarcinoma group and in the squamous-cell carcinoma group, but whether this was real or only apparent and whether we were missing those lesions in the left lower lobe is impossible at this time to determine.

TABLE I. AGE DISTRIBUTION BY DECADES IN 384 CASES OF BRONCHOGENIC LARGE-CELL CARCINOMA

AGES, YEARS	CASES	PER CENT
20-29	3	0.8
30-39	18	4.7
40-49	59	15.4
50-59	179	46.6
60-69	117	30.4
70 OR MORE	8	2.1
TOTAL	384	100.0
MEAN AGE	55.0 YEARS	

In seventy-five of the cases in which resection was performed, bronchoscopy was carried out prior to operation and positive results on biopsy were obtained in thirty-one of these. In all those in which biopsy gave positive results the lesion was shown to be central at the time of resection. These thirty-one cases represented only 41.3 per cent of the cases in which bronchoscopy was performed, but they represented 62 per cent of all the central lesions.

Symptoms present were, in general, those of any type of bronchogenic carcinoma. The five commonest symptoms were cough, loss of weight, pain, hemoptysis, and dyspnea in the following incidence: cough, 80.7 per cent; loss of weight, 54.4 per cent; pain or aching in the chest, 47.9 per cent; blood-streaked sputum or hemorrhage, 44.0 per cent; and dyspnea, 29.5 per cent. The average duration of symptoms from onset to histologic diagnosis was 8.4 months in the cases in which biopsy was done and 6.4 months in the cases in which resection was performed (Table II).

Cytologic examination of the sputum and bronchial secretions for malignant cells was performed in 144 cases and malignant cells were found in 111, or 77.1 per cent. Cytologic examination was judged as inadequate in twenty of the thirty-three cases in which the results were negative, that is, inadequate in that less than three specimens for examination were received in the laboratory. With these excluded, cytologic examination gave positive results in 89.5 per cent of the cases in which an adequate number of specimens were available.

From 1940 through 1944 there were 105 cases, in thirty-four of which exploration was carried out; resection was done in twelve of the thirty-four cases. This gives an exploration rate of 32.4 per cent, a resection rate of 11.4 per cent, and a ratio of resection to exploration of 35.2 per cent. From 1945 through 1949 there were 200 cases, in 116 of which exploration was performed; in 65 of the 116 resection was done. This gives an exploration rate of 58 per cent, a resection rate of 32.5 per cent, and a ratio of resection to exploration of 56

TABLE II. DURATION OF SYMPTOMS IN 384 CASES OF BRONCHOGENIC LARGE-CELL CARCINOMA

DURATION OF SYMPTOMS (MONTHS)	SOURCE OF SPECIMEN	
	BIOPSY (PER CENT OF 297 CASES)	RESECTION (PER CENT OF 87 CASES)
ASYMPTOMATIC	2.4	12.6
1-3	22.9	24.1
4-6	30.0	29.9
7-9	15.2	14.9
10-12	13.8	4.6
13-24	12.8	10.3
25-36	2.7	3.4
MEAN	8.4 MONTHS	6.4 MONTHS

per cent. Apparently the higher resection rate in the latter group was due both to a higher rate of exploration and to a higher rate of resection in the cases in which exploration was done.

In ten cases, reports of roentgenologic examination were not available. Of the remainder, in eight the roentgenograms were reported as normal, and in five they were reported as normal, but the notation was made in the latter cases that the pulmonary fields were obscured by residual oil. In all the remaining cases there were abnormal roentgenologic findings, but it was felt that in 12.5 per cent of these, bronchogenic carcinoma was not suspected. In those in which we felt that bronchogenic carcinoma was not suspected, the diagnosis most often made was pulmonary tuberculosis.

With this cell type of bronchogenic carcinoma there is no grading problem, because by definition all the lesions must be grade 4.

It has already been pointed out in this series of papers that resection of portions of the lung for carcinoma has been rather a recent development in surgery. There were twelve hospital deaths (13.8 per cent) among the eighty-seven patients in this group. Only twenty-six of eighty-seven patients were operated on before 1947, and forty-one, or nearly half, were operated on during 1948 or 1949. It was felt, therefore, that survival rates* should be calculated only on a short-term basis (Table III). Forty-eight and six-tenths per cent of the patients were dead in a year, and 56.4 per cent were dead within two years. A breakdown of the twenty-four patients who lived two or more years following operation is as follows: six patients died in the third year following operation, one in the fourth year, and three in the fifth year; five patients are living in the third year following operation, five in the fourth year, two in the fifth year, and two patients are still living more than ten years following operation. This indicates that prognosis is poor in this type of bronchogenic carcinoma, but perhaps not as bad as that for bronchogenic adenocarcinoma or small-cell carcinoma and not nearly as good as that for squamous-cell carcinoma. The reader will recall that the large-cell type of bronchogenic carcinoma is the one in which the cells are so anaplastic that no more specific type of microscopic diagnosis should be made than that of "high-grade carcinoma, type indeterminate." These, therefore, should logically be the most malignant and carry the poorest prognosis of all types of bronchogenic carcinoma with the exception of the small-cell type. We have taken the surprising survival of some of these patients to mean that a goodly proportion of the lesions actually must be squamous-cell carcinomas which are of such high grade that they cannot be so diagnosed, and yet carry over some of the better prognosis to be expected of the squamous-cell type of bronchogenic carcinoma.

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*The calculations of the survival rates were made by the direct method as described by Berkson and Gage (Proc. Staff Meet., Mayo Clin. 25: 270-286, 1950).

TABLE III. SURVIVAL RATES FOR PATIENTS WITH BRONCHOGENIC LARGE-CELL CARCINOMA FOLLOWING PNEUMONECTOMY OR LOBECTOMY

PERIOD, YEARS	PATIENTS		LIVED BEYOND INDICATED PERIOD	
	TOTAL	TRACED	NUMBER	PER CENT*
1	75	74	38	51.4
2	56	55	24	43.6

*Based on traced patients, inquiry as of Jan. 1, 1951. Included in the one-year group are only those patients operated on one or more years prior to the time of inquiry, that is, operated on in 1949 or earlier; the two-year group includes only those patients operated on in 1948 or earlier. *Hospital mortality is excluded in the calculation of survival rates.*

SUMMARY

The criteria for diagnosis of bronchogenic large-cell carcinoma have been presented, and the microscopic relation to other types of bronchogenic carcinoma reviewed. It was found that 40.2 per cent of the bronchogenic carcinomas in this series were of the large-cell type, and that 14.6 per cent of the patients were females. Age distribution, symptoms, and roentgenologic findings were similar to those in other types of bronchogenic carcinoma. The ratio of central to peripheral lesions was found to be 1.4 to 1 for large-cell carcinoma, as compared with 1.9 to 1 for squamous-cell carcinoma, 27.7 to 1 for small-cell carcinoma, but with 0.54 to 1 for adenocarcinoma. Cytologic diagnosis gave positive results in nearly 90 per cent of adequate examinations. About 55 per cent of the patients who survive resection die within two years. This indicates a poor prognosis in this type of bronchogenic carcinoma, but perhaps the prognosis is not as bad as that for bronchogenic adenocarcinoma or small-cell carcinoma, and not nearly as good as that for squamous-cell carcinoma.

HEMODYNAMICS: STUDIES ON A GROUP OF PATIENTS WHO DEVELOPED COR PULMONALE FOLLOWING THORACOPLASTY

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KYPHOSCOLIOSIS has been recognized as a cause of heart disease for many years. Thoracoplasty has been mentioned as a cause for cor pulmonale, but has received little attention in medical literature. It is the purpose of this paper to report on physiologic observations in a group of five patients who developed cor pulmonale within eight to fifteen years following thoracoplasty. No special effort or case finding was made to detect these cases. They were gleaned from a series of over 650 cardiac catheterizations carried out under the direction of the author.

METHOD

The patients were studied in the basal state with administration of 0.06 Gm. Nembutal one hour before catheterization. The cardiac outputs, done in duplicate, were determined by the direct Fick principle. The oxygen consumption of the patient was determined by measuring the volume of inspired air on a specially designed gas meter,[1] and the oxygen content of the expired air and of the room air was determined by means of a Pauling oxygen analyzer. The blood oxygen contents were determined by the spectrophotometric method of Hickam and Fraser.[2] Pressures in the femoral artery and from the right side of the heart, as well as the electrocardiograph and phase of respiration, were recorded on a six-channel oscillograph by means of Statham gauges and strain amplifiers.[3]

CASE REPORTS

A clinical résumé of the five cases studied is as follows:

CASE 1.—B. G., aged 24 years, female. Ten-rib left thoracoplasty fourteen years ago for tuberculosis. The patient did well until one month ago when she contracted a cold. Physical examination: vital capacity, 40 per cent of normal; P_2 greater than A_2; P_2 shock was felt. Right ventricle was active. Regular sinus rhythm. Right axis deviation in the electrocardiogram and pulmonale P waves. On fluoroscopy there was a large pulmonary artery.

CASE 2.—C. B., aged 39 years, male. Eight-rib thoracoplasty on left in 1937, for tuberculosis. Upper six ribs and corresponding second stage in November, 1937. *Third stage* consisted of transverse processes in 1938. History of cardiac failure for three weeks prior to admission. Summary of cardiac findings showed P_2 to be greater than A_2. There were no murmurs, regular sinus rhythm. The electrocardiogram showed the heart was verticle with tendency to right axis deviation, and peaked P waves, Leads II and III. Fluoroscopy of the heart was unsatisfactory due to distortion caused by preceding operations. Vital capacity was 34 per cent of normal.

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CASE 3.—M. V., aged 33 years, female. First stage, left thoracoplasty in October, 1941, with vital capacity 36 per cent of normal. Second stage, posterolateral in November, 1941—anterolateral cartilages 1, 2, and 3, and anterior segments of 2, 3, and 4, and peaked P waves in Leads II and III. Vital capacity was 30 per cent of normal. Third stage, eight-rib anterolateral in January, 1942, with vital capacity 30 per cent of normal. Cardiac findings: loud P_2, regular sinus rhythm. The electrocardiogram showed large P waves, right axis deviation. Prominent pulmonary artery on fluoroscopy.

CASE 4.—M. C., aged 41 years, male. Seven-rib thoracoplasty in December, 1939. Vital capacity was 55 per cent of normal. Cardiac findings: P_2 greater than A_2, regular sinus rhythm, no murmurs. The electrocardiogram showed prominent P waves, Leads II and III. Fluoroscopy unable to evaluate due to distortion of chest wall. Vital capacity in May, 1940, was 51 per cent of normal. At time of study vital capacity was 44 per cent.

CASE 5.—A. R., aged 50 years, male. Tuberculosis with emphysema since 1933. Oleothorax followed by thoracoplasty and phrenic crush in 1936. First stage on March 4, 1936, vital capacity was 52 per cent of normal. Second stage on March 28, 1936; vital capacity was 28 per cent. Third stage on April 18, 1936; vital capacity was 30 per cent. The three operations resulted in the removal of varying lengths of ribs one to ten and the corresponding transverse processes. Four months before study he began to develop shortness of breath. Cardiac findings: A_2 greater than P_2, regular sinus rhythm, no murmurs. It was difficult to interpret heart size due to chest deformity. The electrocardiogram showed left axis deviation. Semi-horizontal heart, moderate right and left ventricular hypertrophy. On fluoroscopic examination the pulmonary arteries were prominent, and the right ventricle appeared slightly enlarged.

RESULTS

Table I summarizes the results of the physiological studies in these five cases. All had marked elevation in pulmonary artery pressure, and pulmonary resistance was markedly increased. The pulmonary diastolic pressures were markedly elevated from 100 to 500 per cent above normal. The above changes are consistent with those found in another group of patients studied who had cor pulmonale from a variety of causes.[4]

Pulmonary capillary pressures were measured in two patients, and were found within the range of normal. Dexter[5] was the first to show that patients with cor pulmonale had normal pulmonary capillary pressures. This has been confirmed by us.

The problem of cardiac output in patients with cor pulmonale still is not clear. We have observed patients with cor pulmonale in failure without elevated cardiac outputs, and again we have observed elevated outputs in patients with cor pulmonale and congestive failure.[4] It would appear that the degree of anoxia and the amount of low-grade chronic infection that is present in the lungs of this type of patient determine, in a sense, the output gradient.

DISCUSSION

It is of interest why patients with thoracoplasty should develop cor pulmonale, if the remaining lung has no disease. In these five patients the disease was limited to one lung.

Ackerman and Kasuga,[6] in 1941, conducted a survey of the various changes in pulmonary tuberculosis which tended to affect the right side of the heart with hypertrophy of the ventricular wall as a result. In this connection they stressed that chronic productive tuberculosis with emphysema, marked pneumothorax

TABLE I. SUMMARY OF HEMODYNAMICS IN A GROUP OF FIVE PATIENTS WHO DEVELOPED COR PULMONALE FOLLOWING THORACOPLASTY*

NAME	PULMONARY ARTERY PRESSURE		FEMORAL ARTERY PRESSURE		RIGHT VENTRICLE	RIGHT AURICLE	PULMONARY CAPILLARY PRESSURE	CARDIAC INDEX	CARDIAC OUTPUT	SYSTEM RESISTANCE (DYNES/CM.^{5})	PULMONARY RESISTANCE (DYNES/CM.^{5})	HEART RATE	STROKE VOLUME (C.C.)
	MM.HG.	MEAN	MM.HG.	MEAN									
V., M. 33 W., F.	90/52	68	96/67	75	96/10	14/3		3.34 L.	4.65 L.	1795	1621	100	46
B., C. 39 W., M.	34/20	24	210/125	145	30/2	5/3	3.5 M.P.	3.82 L.	6.29 L.	3150	510	107	59
G., B. 24 W., F.	57/29	38	93/63	80	53/0	4.3/−2		4.34 L.	5.02 L.	1480	705	100	50
R., A. 50 B., M.	41/20	28	116/63	87	37/0	4.4/1	5 M.P.	2.60 L.	4.45 L.	2680	857	73	61
M., C. 41 B., M.	38/23	29	119/84	103	40/2	6/3.5		5.16 L.	10.92 L.	1590	445	91	120

*Note: Pulmonary capillary pressure was measured in two of the patients in this group and was found in the range of normal.

of long duration, and thickened pleura, sometimes gave rise to the right side hypertrophy. Finally they claimed thoracoplasty may be a factor in the production of the right-sided ventricular hypertrophy.

Higgins,[7] in 1944, in a series of 600 autopsy cases of pulmonary tuberculosis, observed a slight increase in the weight of the right ventricle. He was unable to find a correlation between right-side hypertrophy and individual collapse therapy, although he did observe a correlation with the duration of the disease.

Griggs, Coggin, and Evans[8] studied a series of 18,000 autopsied cases with a view to ascertaining the extent to which chronic pulmonary diseases led to cor pulmonale. They found the wall of the right ventricle at least 0.5 cm. thick in more than 50 per cent of the cases of emphysema, but in only 3.7 per cent of the 1,470 cases of pulmonary tuberculosis.

However, the great numerical superiority of the latter cases meant that they found hypertrophy of the right ventricle more often in cases of pulmonary tuberculosis than in any other disease of the lungs.

Gibbon, Hopkinson, and Churchill,[9] in 1932, showed in animal experiments that signs of cor pulmonale did not appear until at least 60 per cent of the blood flow through the pulmonary artery had been cut off. Recently, we have shown that although there is a transitory marked rise at the time of tying off of the right or left pulmonary artery during a pneumonectomy, this pressure soon begins to return to a normal level by compensation of the remaining vascular bed, and usually by the time this operation is completed, the pressure had returned to preoperative levels.[10]

Cournand[11] has also shown in studies that the pressures in the pulmonary artery, after patients have had pneumonectomy, are still in the normal range if they had no disease in the remaining lung. He has measured pressures in the pulmonary artery several years after pneumonectomies and has found normal values.

Bruce[12] reported on two cases of thoracoplasty who developed cor pulmonale, proved at autopsy. He emphasized the fact that after operation the thoracoplastic patient often showed a striking gross anatomic resemblance to the kyphoscoliotic patient, as usually a deformity of the chest develops in connection with the operation, and this deformity involves the spine and gives rise to a more or less pronounced spinal curvature, with the convexity to the operated side. Bruce also pointed out that in spontaneous thoracic deformities, scoliosis, with the convexity on the right side, is for some reason more common than scoliosis with left side convexity. It has been claimed that cardic pulmonary insufficiency, especially, tends to appear when the deformity is accompanied by right-sided compensatory compressions of the left side of the thorax which, in turn, has a specific effect on the heart and the conditions under which it functions. Three of our five cases had left-sided thoracoplasties.

Certain it is that thoracoplasty has a great deal to offer the selected case of tuberculosis. However, one of the complications that a patient with a thoracoplastry may encounter is cor pulmonale. It would also seem difficult to predict the patient who will develop cor pulmonale following thoracoplasty. Certainly it is, the more extensive the collapse the greater the chance of developing cor pulmonale.

SUMMARY

Five patients with a thoracoplasty who developed cor pulmonale as a complication are reported with physiological studies. All five patients studied had typical findings, on intracardiac catheterization, of cor pulmonale.

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SOLITARY PULMONARY ADENOMA (FOCAL PULMONARY ADENOMATOSIS): A THREE-YEAR FOLLOW-UP AFTER RESECTION

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UNTIL recently, the diagnosis of pulmonary adenomatosis was made only at autopsy. However, an increasing awareness and interest in this entity has resulted in a number of instances of clinical diagnoses and, in a few, of treatment by surgical resection.

From 1896, when Livingood[1] described the multicentric nature of spontaneous lung tumors in mice, there has been controversy as to the origin of the tumors of pulmonary adenomatosis and of alveolar cell carcinomas of the lung. However, there has been fair agreement on the multicentric nature of these tumors. Delarue and Graham[2] emphasized this point and cautioned against false optimism following resection. They reported a case with an uneventful recovery following right lower lobe resection in June, 1943. The patient remained well until the fall of 1946, when he developed a productive cough, weight loss, and pain in the right chest and arm. In December, 1947, the remainder of the right lung was resected, but there had been invasion of local tissues so that, despite the fact that the left lung was still clear in September, 1948, the authors felt the prognosis was guarded.

More recently, Osserman and Neuhof[3] reported a five year follow-up without recurrence in a 44-year-old woman who had had a left lower lobe resection for localized "mucocellular papillary adenocarcinoma."

Both the case reported by Delarue and Graham[2] and that reported by Osserman and Neuhof[3] were symptomatic.

We believe the following case worthy of reporting because of the youth of the patient, the asymptomatic course, the long preoperative roentgenographic observation, and the lack of recurrence over the three-year postoperative period.

CASE REPORT

M. C., a 16-year-old applicant for a summer job at the Philadelphia Navy Yard, had a pre-employment 46 mm. photofluorogram on June 29, 1944 (*Fig. 1*), which revealed a small rounded mass with a central area of rarefaction at the left base. During this period in the survey work of the Philadelphia Tuberculosis and Health Association no routine follow-up was maintained, reliance having been placed in the follow-up by the plant physicians. In the spring of 1945, one of the authors, in taking over the survey project, came across the photofluorogram in a casual study of *previous significant films*. The young man had been rejected for employment because of the findings and the family had moved, so he could not be found until July, 1945, when roentgenogram revealed no change (Figs. 2 and 3). He was a

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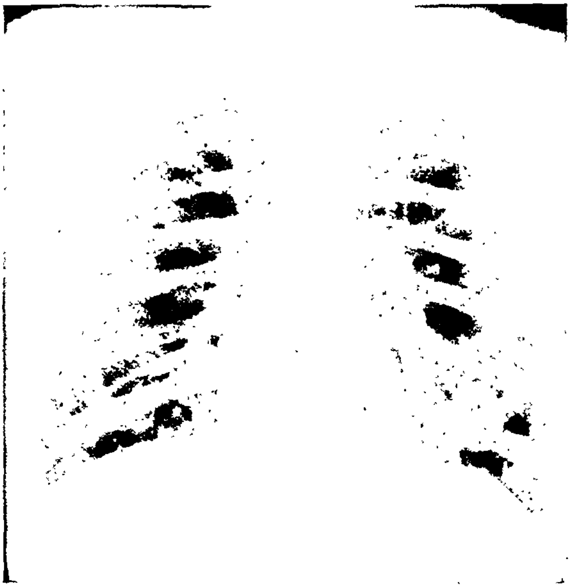


Fig. 1.—June 29, 1944. 70 mm. photofluorogram revealed rounded mass with central area of rarefaction at left base.

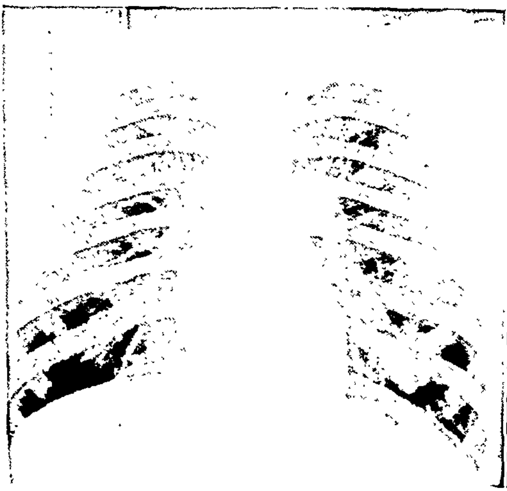


Fig. 2.

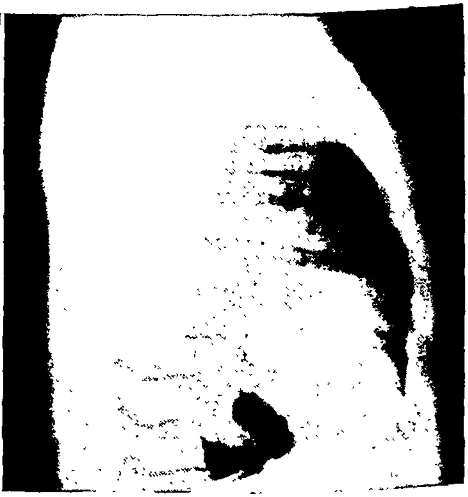


Fig. 3.

Fig. 2.—July 11, 1945. 14 × 17 retake revealed no change.
Fig. 3.—July 11, 1945. Lateral reveals mass in left lower lobe.

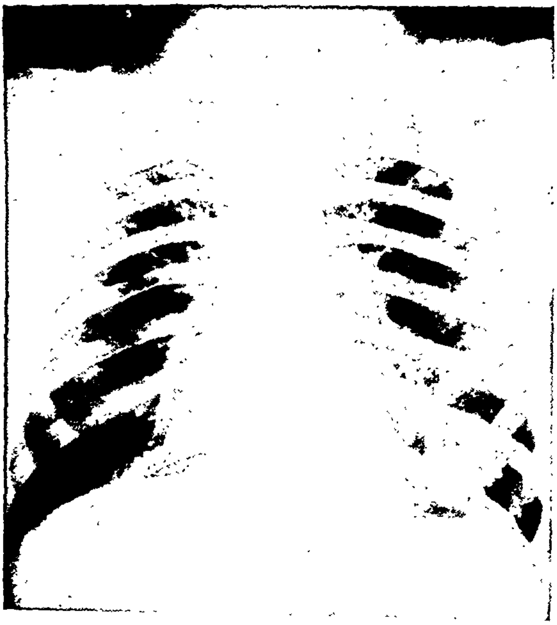
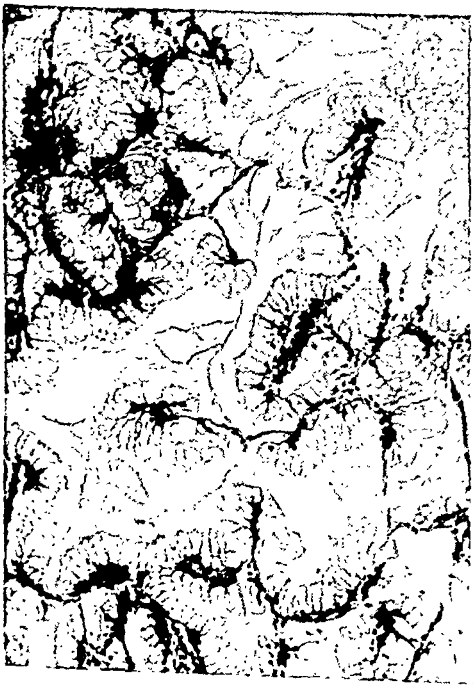


Fig. 4.—Sept. 15, 1947. Lesion has definitely increased in size.



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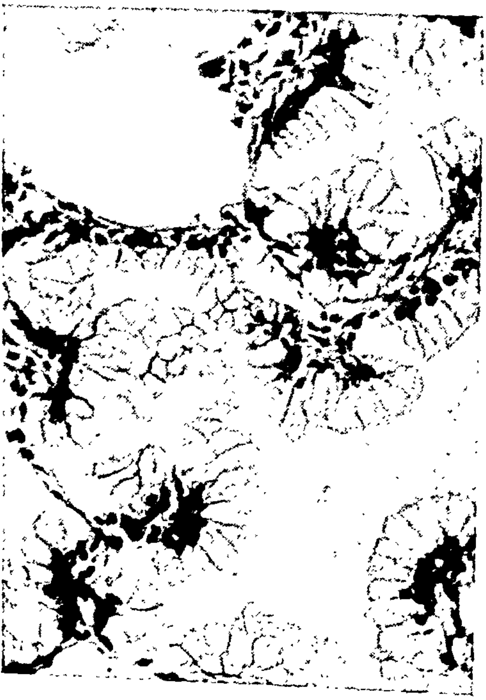

Fig. 6.

Fig. 5.—Photomicrograph, low magnification, showing alveolar arrangement of tumor cells.

Fig. 6.—Photomicrograph, high magnification, showing mucin-secreting cells and their nonmalignant character.

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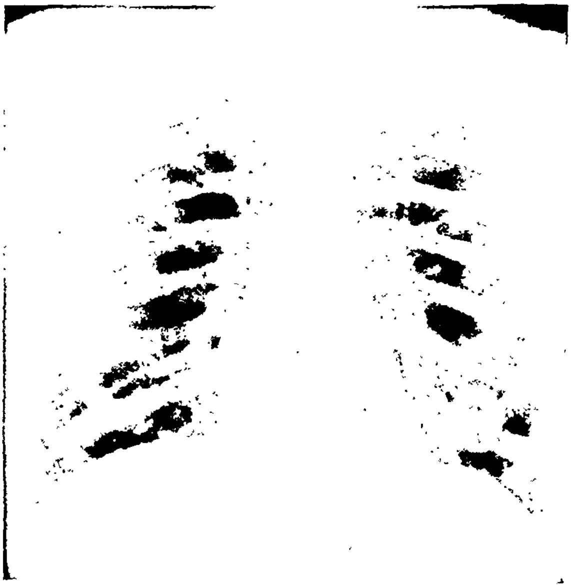

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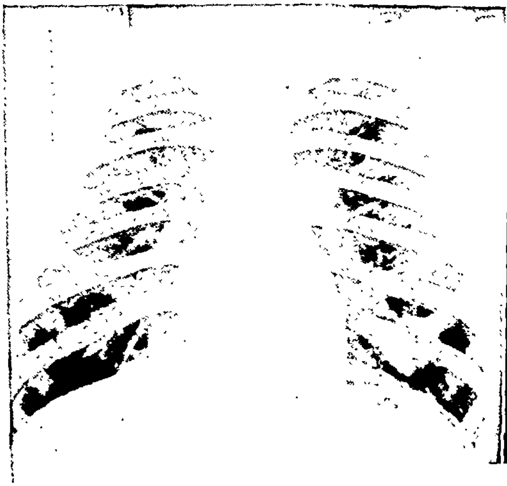

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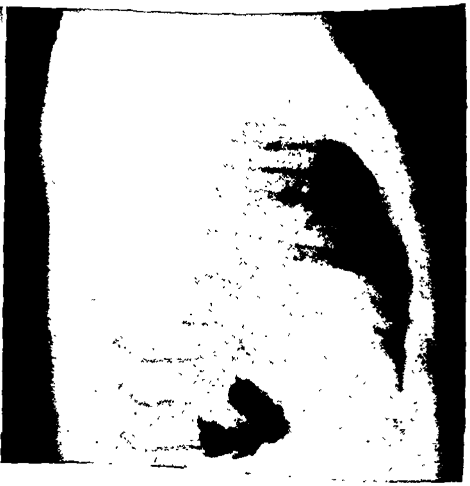

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was noncontributory. Laboratory tests were normal except for a white cell count of 13,000 with 88 per cent neutrophilic leucocytes. At operation, Nov. 4, 1947, a tumor of approximately 7 cm. diameter was found in the left lower lobe, near the interlobar fissure directly beneath the pleura. The entire mass was removed with a margin of lung tissue for biopsy.

The surgical specimen was a flat piece of tissue covered on one surface by pleura measuring approximately 5 cm. in diameter. The specimen was about 3 cm. thick. In its substance there was a spheroid mass which was rather soft and had a pinkish color. It was in close proximity to the pleura along one surface; elsewhere it was poorly delineated, merging imperceptibly into normal lung tissue. When it was sectioned an astonishing quantity of clear, sticky exudate emerged. It had the appearance and consistency of rather thin egg white. The central portion of the mass was cystic, containing more of this clear exudate.

Blocks were fixed in formalin solution and sectioned. They presented a bizarre microscopic appearance. The entire mass was composed of alveolar structures which had the size and pattern of pulmonary alveoli without alveolar ducts (Fig. 5). The walls of these structures looked like alveolar partitions, and this resemblance was enhanced by the presence of several capillary blood channels. Most of the structures were lined by a single layer of tall, narrow, cylindrical cells measuring from 75 to 100 micra in length and 10 to 20 micra in width. The nuclei, about 10 micra in diameter, were uniform, darkly chromatinized and situated at the extreme bases of the cells. They lacked the appearance which is associated with a malignant behavior. The cytoplasm stained poorly and under high magnification was seen to consist of various sized globules of mucin. The cells were not ciliated. The alveolar lumen was filled with the same mucinous substance which formed the cytoplasm (Fig. 6). It was impossible to tell whether the free mucin was formed by secretion or by sloughing and disintegration of the alveolar lining cells, though there was a considerable amount of the latter leaving many of the alveolar partitions completely bare (Fig. 7).

The postoperative course was uneventful. Focal adenomatosis was the final diagnosis. No recurrence has been demonstrable (Fig. 8). It is noteworthy that no change occurred in this lesion until more than three years after it was first found.

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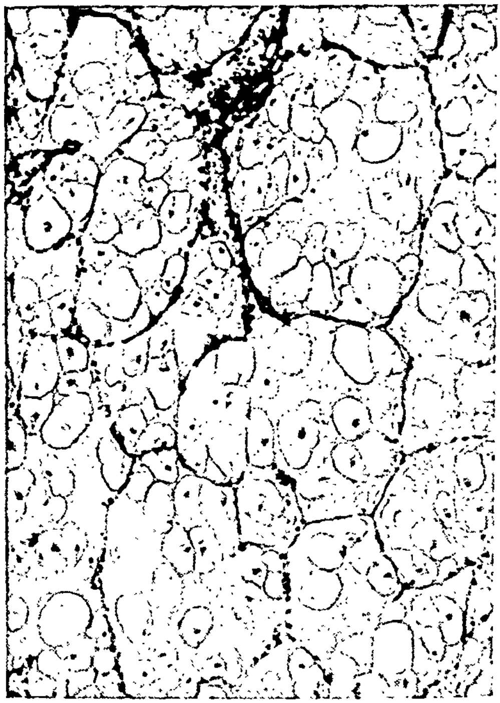

Fig. 7.—Photomicrograph of tumor, showing sloughing and disintegration of tumor cells to form sticky exudate.

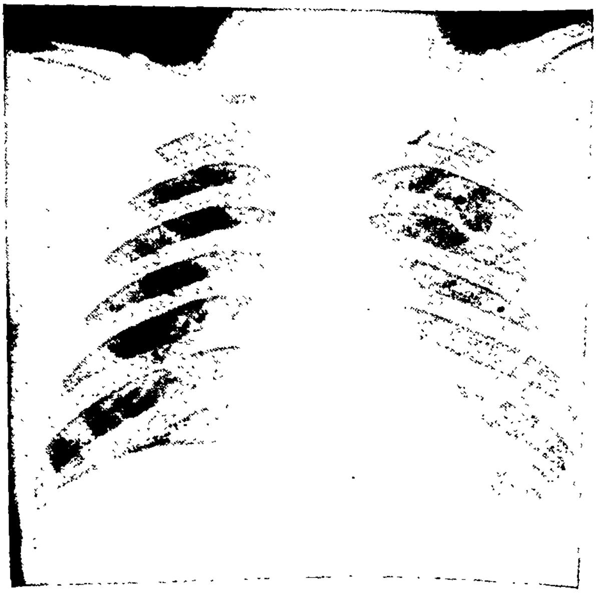

Fig. 8.—Aug. 1, 1950. No recurrence visible.

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THE INFLUENCE OF STREPTOMYCIN RESISTANCE UPON THE SUCCESS OF CAVERNOSTOMY FOR THORACOPLASTY FAILURE*

JAMES D. MURPHY, M.D., AND H. V. SWINDELL, M.D., OTEEN, N. C.

THE August, 1950, issue of THE JOURNAL OF THORACIC SURGERY contained a report[1] of a series of 23 thoracoplasty failures who were treated at the Veterans Administration Hospital, Oteen, North Carolina, with an Eloesser type cavernostomy. These patients were operated upon in late 1946 and early 1947. Streptomycin was not in common use at that time. Only two of the patients in the series had received the drug prior to the time when an attempt was made to close the cavernostomy sinus. A negative sputum and closure of cavity were obtained in 81.2 per cent of sixteen patients. Streptomycin was used as an adjunct at the time of closure of the residual sinus. The results impressed us so favorably that we subsequently chose the operation in place of resection in a number of instances.

As the therapeutic application of streptomycin in tuberculosis has become more widespread, we rarely receive patients for treatment who have not had one or more courses prior to consideration for surgery. The patients reported in the original study have continued to do well. The series has now been expanded to a total of forty-one patients. Twenty-five of these had received streptomycin prior to the open cavity drainage.

More than 3,000 articles have appeared in the literature since the discovery of streptomycin in 1944 by Schatz, Bugie, and Waksman,[2] and the report by Feldman and Hinshaw[3] concerning its activity againt *M. tuberculosis.* Many of these have dealt with the application of the drug to the surgery of tuberculosis.

The protective action of streptomycin against spreads, bronchopleural fistula, and empyema following thoracic surgical procedures has been repeatedly emphasized. As early as 1948, however, evidence began to appear indicating that the unrestricted use of streptomycin in thoracic surgery was useless, and possibly harmful. At that time the Veterans Administration discontinued the routine use of streptomycin as a prophylactic agent during thoracoplasty for tuberculosis.

Since then, except in the several "Transactions of the Streptomycin Conferences" little has been written, concerning the effects of previous streptomycin treatment and the development of resistance upon the results of surgery for tuberculosis. During the Sixth Streptomycin Conference in 1948, Coleman and Bunch[4] asked the question, "Is Pulmonary Resection Safe in Patients Previously Treated with Streptomycin?" After citing two unfavorable cases, their con-

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clusion was to the contrary. Early in 1949, Murphy and Seiler[5] studied the question of bronchopleural fistulas following pulmonary resection in patients who had been treated with streptomycin prior to their resection course. An incidence of 25 per cent of fistula development was encountered in the previously treated patients, as compared with 5 per cent in those who had received no streptomycin previous to resection.

Later in 1949, Murphy, Seiler, and Walkup[6] discussed complications following resections in patients previously treated with streptomycin. They tabulated the incidence of spreads, bronchopleural fistulas, late reactivation, empyema, wound infection, and the mortality rate, and found that in each case the rate was higher in the previously treated than in the untreated patients.

The following year, 1950, Murphy and Walkup[7] again stated that the incidence of complications was higher in a streptomycin resistant group than in a nonresistant group of previously reported patients who had been given a thoracoplasty for tuberculosis. The work included the results after resections or cavernostomies in the thoracoplasty failures.

The "Quarterly Progress Reports of the Veterans Administration Streptomycin Committees"[8] in July, 1950, reviewed the statistics of the surgical staff of the Veterans Administration Hospital, in Minneapolis, Minn. Results after sixty-four resections performed at the Minneapolis Hospital were compared with those after 630 resections performed in other Veterans Administration Hospitals. It was found that in a group of 408 lobectomies there were marked differences in the rate of sputum conversion and incidence of postoperative complications between streptomycin resistant and previously nontreated groups. Fifty per cent higher incidence of sputum conversion was found in the previously untreated group. They found three times as many postoperative spreads, fifteen times greater incidence of bronchopleural fistulas, and twenty-five times greater incidence of empyema in the resistant group. There were comparable figures in the pneumonectomy group. In the sixty-four resections performed at their own hospital, similar results were obtained.

The earlier studies quoted have given considerable evidence as to the harmful effects of streptomycin resistance upon the results of excisional surgery and thoracoplasty for tuberculosis. Since our recent results following cavernostomy for thoracoplasty failure have been discouraging, we decided to analyze the series and attempt to determine if streptomycin resistance was a factor.

This paper compares the results following cavernostomy in patients who have not had previous streptomycin treatment with those who have had such treatment.

The indications for operation and the operative technique as given by Murphy and Koontz in their original description have been constant throughout the entire series.

Table I demonstrates the influence of streptomycin on cavity closure.

Cavities Opened.—Forty-one cavities in an equal number of patients have been opened by an Eloesser type cavernostomy. Sixteen of these patients had not received streptomycin therapy previously. Twenty-five patients had been previously treated with streptomycin, and twenty-three of these were known to be resistant.

TABLE I. INFLUENCE OF STREPTOMYCIN ON CAVITY CLOSURE

	CAVITIES OPENED	CAVITIES CLOSED		PER CENT CLOSURE	AVERAGE IN WEEKS OF DRAINAGE
		PRIMARY	SECONDARY		
Without Previous Streptomycin	16	8	5	81.2	18.6
With Previous Streptomycin	25	3	4	28.0	31.3

Cavities Closed.—Thirteen of the sixteen patients who had not received previous streptomycin therapy, or 81.2 per cent, obtained a successful eradication of the cavity by cavernostomy alone. Eight of these healed primarily following surgical closure and five healed secondarily, exhibiting cutaneous fistulas for varying lengths of time. Of the three patients classified as cavernostomy failures, two eventually underwent resection. One of these failures died (to be discussed later). A negative sputum was obtained in both of the patients who had a resection. One of these developed a postoperative bronchopleural fistula. Only 7, or 28.0 per cent, of the 25 patients who had been previously treated with streptomycin could be classified as successful cavernostomies. Three of these healed primarily following surgical closure, and four healed secondarily. Ten of the remaining eighteen patients have persistent cavities.

An attempt at surgical closure failed in eight patients. Ten never fulfilled the criteria for surgical closure. Resection was performed in five patients in whom surgical closure was unsuccessful. Three of these healed and a negative sputum was obtained. Two of the resection patients died.

Average Duration of Drainage, in Weeks.—This column in Table I denotes the number of weeks elapsing from the time the cavity was opened until it was felt that surgical closure of the wound was feasible. Closure was attempted when the cavity was no longer visible on planigraphic roentgenographic studies, and no longer palpable on digital examination through the cavity stoma. In most instances, also, the tract was epithelized and the drainage reduced to a scanty mucoid material. Smears and cultures of the sinus drainage were usually found to be negative for acid-fast bacilli. The figure of 18.6 weeks represents the average drainage time of the thirteen successful cavernostomies performed in patients who had not been previously treated with streptomycin. The longest drainage time in this group was twenty-eight weeks, and the shortest six weeks.

The average drainage time of the seven successful cavernostomies performed in the group who had previously been treated with streptomycin was 31.3 weeks. The longest time was thirty-six weeks, and the shortest 20 weeks. It is of interest to note that the shortest drainage time in the previously treated group was longer than the average drainage time for the previously untreated group. The ten previously treated patients, having persistent cavities, have continued to drain an average of 50.4 weeks.

Table II demonstrates the influence of streptomycin on sputum conversion and complications.

Prior Positive Sputum.—Fifteen of the sixteen previously untreated cases had a postive sputum prior to the time of cavernostomy. Twenty-three of the twenty-five previously treated cases had positive sputum preoperatively.

TABLE II. INFLUENCE OF STREPTOMYCIN ON SPUTUM CONVERSION AND COMPLICATIONS

	PRIOR POSITIVE SPUTUM	SPUTUM CONVERSION		SPREADS		WOUND INFECTIONS		DEATHS	
		NO.	PER CENT	NO.	PER CENT	NO.	PER CENT	EARLY	LATE
Without Previous Streptomycin	15	13	86.6	1	6.2	1	6.2	1	0
With Previous Streptomycin	23	10	43.5	9	36.0	4	16.0	2	3

Sputum Conversion.—Thirteen of the fifteen previously untreated patients who had preoperative positive sputum, or 86.6 per cent, obtained a sputum negative by culture. Only ten of the twenty-three previously treated cases with positive sputum, or 43.5 per cent, obtained a conversion of sputum. The patients with no prior treatment showed approximately 50 per cent greater incidence of sputum conversion.

Spreads.—In the group of sixteen streptomycin "virgins" undergoing cavernostomy, there was a contralateral spread in the patient who developed traumatic pneumothorax and died on the eleventh postoperative day. Among the group of twenty-five previously treated patients there were nine spreads, a total of 36.0 per cent.

Wound Infections.—The question of wound infection is hard to evaluate in cavernostomies, since all of the wounds, due to the nature of the operation, show some degree of infection. In this series only those patients in whom there was a definite wound breakdown or progression of the infection beyond the operative area were classified as having an infection. In the group that had been previously treated with streptomycin there were four, or 16.0 per cent, who showed a definite wound infection.

Deaths.—In the group not receiving previous streptomycin, there was one early death. This occurred eleven days following the second stage, at which time a traumatic pneumothorax was incurred. In the previously treated group there were two early and three late deaths. One of the late deaths occurred after discharge from the hospital, and the other two followed resection for cavernostomy failure. One of the early deaths followed the opening of the cavity, at which time a spinal foramen was entered during the course of needle exploration. Tuberculous meningitis resulted. The other early death resulted from a massive bilateral caseous spread following the cavernostomy closure.

CONCLUSIONS

1. A study of forty-one consecutive cases undergoing cavernostomy for post-thoracoplasty residual cavity reveals that: (a) Sixteen of these had not received streptomycin prior to the surgery, and twenty-five had been previously treated with streptomycin. Twenty-three were resistant to over 10 micrograms per milliliter prior to cavernostomy closure. (b) The operation was more satisfactory and the incidence of sputum conversion was much higher in the previously untreated patients. (c) The incidence of complications was much higher in patients with resistant bacilli than in those who had received no streptomycin prior to closure of their cavities.

2. The indiscriminate use of streptomycin in the treatment of tuberculosis should be condemned.

3. A cooperative medical and surgical study of each candidate should be made prior to initiation of therapy so that the beneficial effects of streptomycin could be given the patient when his needs are most urgent.

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Original Communications

BRONCHOGRAPHY AS AN AID IN PLANNING SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

With a Preliminary Note on Pulmonary Angiography

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A SERIES of lung specimens were studied by injecting radiopaque material into the bronchi and pulmonary arteries and comparing the roentgenographic findings with those of bronchograms in living patients. The interpretation of bronchograms on this basis has been an aid in planning the surgical treatment for certain cases of pulmonary tuberculosis. In this study, which was begun by one of us (J. G.) a little over eight years ago, the bronchial trees and pulmonary arterial system of removed lung specimens were injected with radiopaque material under fluoroscopic control. A variety of radiopaque materials such as bismuth and the iodides were tried, including watery solutions with and without acacia. It was finally decided that an iodized oily solution, as used in bronchography, was the most satisfactory for this purpose. The resultant bronchograms and arteriograms were then correlated with the pathologic changes. It was noted that where there were structural alterations of lung parenchyma due to pulmonary tuberculosis, there were also consistent changes in both the bronchograms and the pulmonary arteriograms.

The lesions present in the lungs of this series of cases are of two apparently distinct types. In the one type, of which Case 7 is an example, the lesion is essentially limited to the large or small bronchi or both, and the surrounding alveolar structure is normal, except for scattered small, isolated scars. The bronchial lesions consist usually of a thin zone of dense collagen deposition beneath the intact epithelium, in the location of the smooth muscle of the wall. The muscle fibers are largely obliterated. In some bronchi of cases showing this lesion, there is seen more or less extensive mucosal ulcera-

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From the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.

Read (by title) at the Thirtieth Annual Meeting of the American Association for Thoracic Surgery, Denver, Colorado, April 15-18, 1950.

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tion with epithelioid and caseous tubercles in the underlying tissue. Presumably, this indicates the mechanism by which the more common lesion of peribronchial fibrosis has developed.

In the other type, exemplified by Case 9, virtually the entire alveolar architecture is replaced by diffuse scarring with foci of caseation or cavity formation. Many of the smaller bronchi and pulmonary vessels are completely effaced. The remaining bronchi course through the wide diffuse scars, are tortuous and usually dilated, but sometimes narrowed. The mucosa is finely serrated. The larger pulmonary vessels associated with these bronchi are patent, but their smaller branches are obscured in the scars and caseous areas, as are the smaller bronchi.

Many lungs show both types of lesion; areas of diffuse scar tissue traversed by tortuous serrated bronchi; and in other areas, normal or emphysematous alveolar structure, with localized peribronchial lesions. Case 8 is an example of a case showing both types. In this instance a recent bronchogenic dissemination had occurred in the lower portion of the left upper lobe. The bronchi show chiefly epithelial ulceration. Some of the tubercles in the bronchial wall project into the lumen. Vascular changes are not apparent.

It appears that the peribronchial fibrosis is responsible for the poor arborization seen in the films of the injected lungs. The involved bronchi show slight serration of the lumen similar to that seen in the bronchograms. In some areas smaller bronchi and bronchioles are seen throughout the lung and are uniformly patent. The orifices of these channels from the larger bronchi are apparently sufficiently narrowed by the peribronchial fibrosis to inhibit the flow of the iodized oil. The generalized emphysema, and the trapping of air noted during life may perhaps be attributed to the same mechanism.

In the normal lung the pattern of the bronchial tree and pulmonary arterial system were similar and paralleled each other in an orderly fashion. There was seen the graceful, delicate arborization with progressive diminution in size of the branches of both systems (Cases 1, 2, and 3). Where the lung contained well-established foci of disease, whether small or very extensive, both bronchi and pulmonary arteries suffered in alteration or obliteration (Cases 4 and 5). The bronchographic patterns observed in these specimens were essentially of two types, and are exemplified in Cases 6, 7, 8, and 9. (1) In extensively damaged parenchyma the proximal larger bronchi were dilated and had irregular, serrated margins. They were somewhat crowded together and stopped rather abruptly so that there was no filling in the distal portion of the bronchial tree.* In these removed specimens cavities filled readily. The pulmonary arteriograms of the areas showing the above bronchial changes revealed a marked paucity of patent arteries. (2) In areas with relatively recent or less extensive disease, such as in bronchogenic spread, the abnormality of the bronchial pattern was less obvious. Some of the bronchioles did not fill at all, while others, even though they filled out to the periphery, were somewhat narrowed, and their fine lacy branches were markedly decreased in number. The larger bronchi did or did not appear normal, depending upon the extent of parenchymal involvement. The injected pulmonary arteries of recent disease still showed patency in some areas, and in others obliteration was seen as in older diseased areas.

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*How this differs from bronchograms of nontuberculous pyogenic bronchiectasis is the subject of another communication.

Following this study of removed lung specimens a variety of conditions was studied by bronchography (Cases 10 to 25) in patients known to have pulmonary

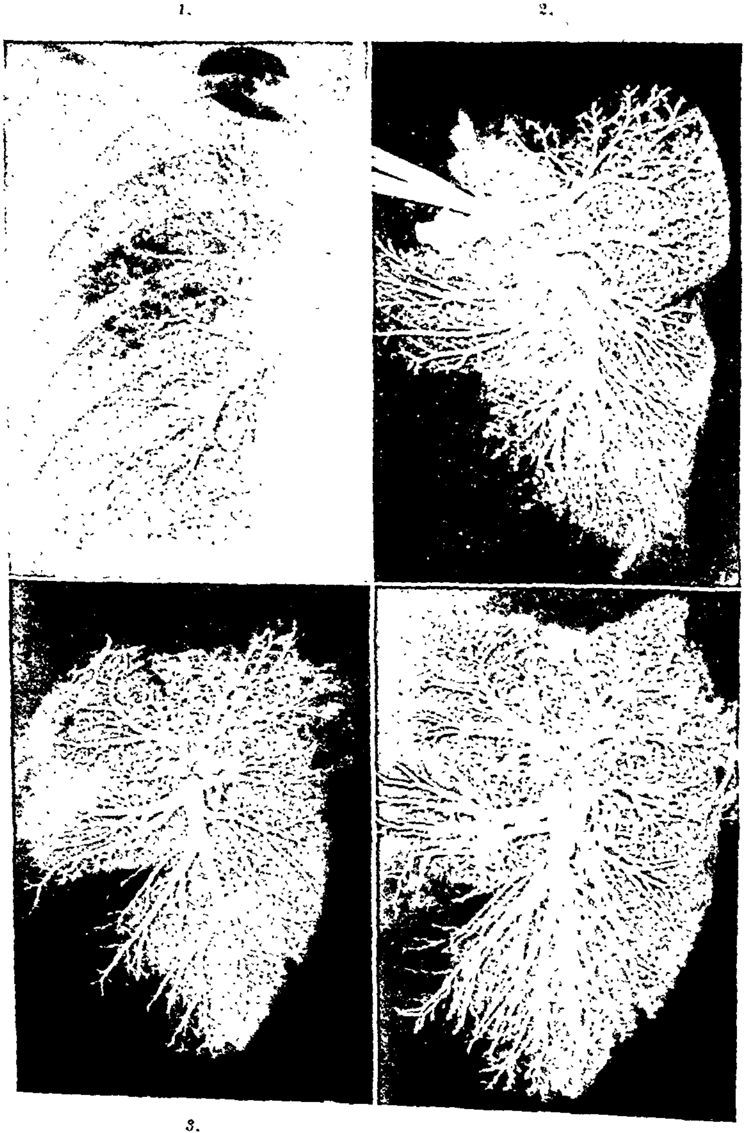


Fig. 1.—*1*, (*Case 1*) Normal posteroanterior bronchogram. *2*, (*Case 2*) Normal pulmonary arteriogram by injection of lung specimen. *3*, (*Case 3*) Normal pulmonary arteriogram by injection of lung specimen. *4*, (*Case 3*) Same lung with additional injection of bronchial tree showing superimposition of both patterns.

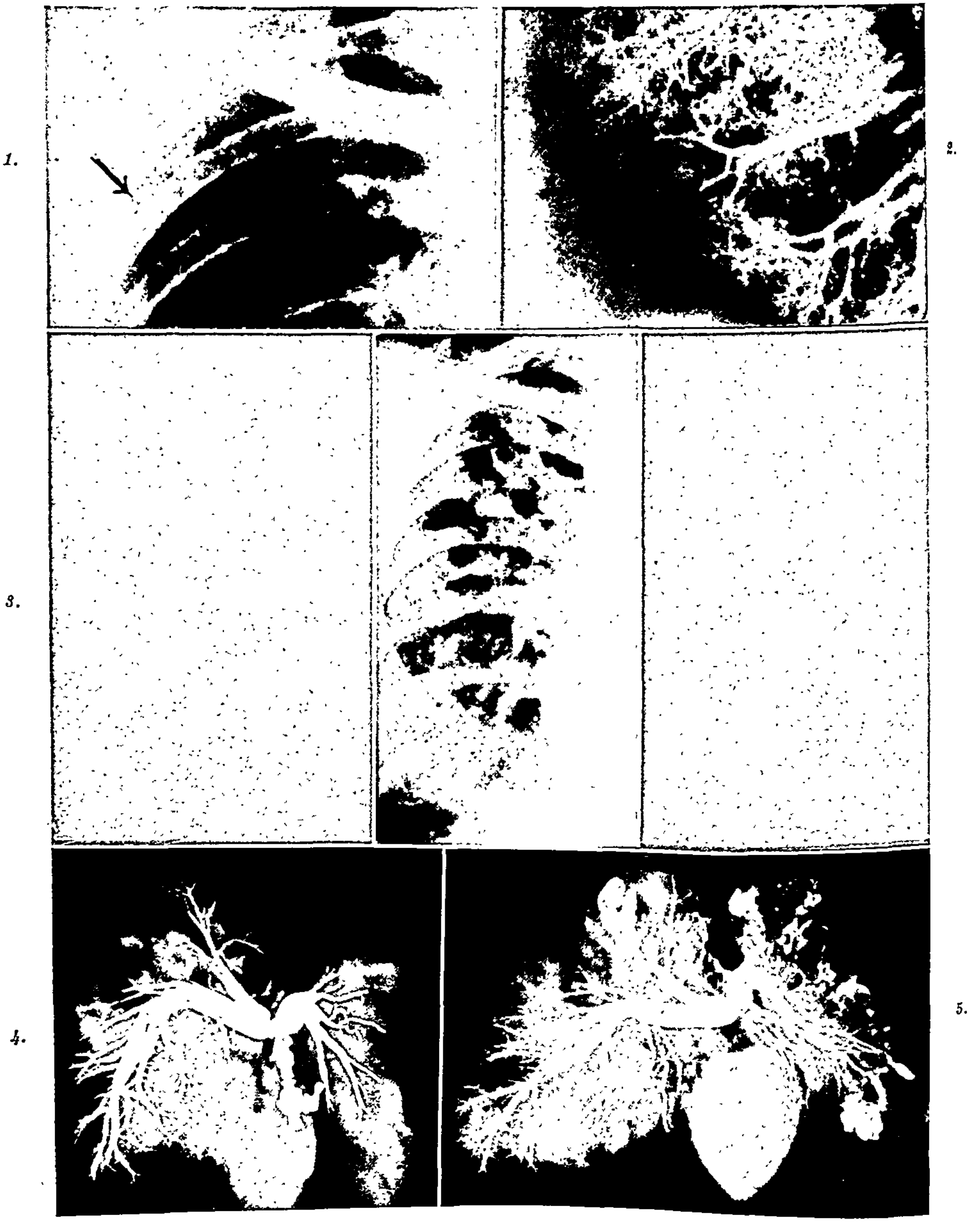


Fig. 2.—*1*, (*Case 4*) Posteroanterior roentgenogram showing shadow of small parenchymal tuberculous focus. *2*, (*Case 4*) Roentgenogram of injected lung specimen showing absence of filling of both pulmonary arterial branches and small bronchi in region of a small parenchymal lesion (by injection of both systems of lung specimen). *3*, (*Case 5*) Posteroanterior roentgenogram showing extensive changes on the right. Thoracoplasty on left not shown. *4*, (*Case 5*) Roentgenogram after pulmonary arterial injection of both lungs in postmortem heart-lung specimen showing marked paucity of arterial pattern. *5*, (*Case 5*) Same specimen with additional bronchial tree injection showing similar poor pattern and filling of cavities.

tuberculosis, and the following observations were made. The bronchographic pattern was altered in the presence of parenchymal disease, and again the two types of changes were commonly noted. With small individual areas of parenchymal involvement the only bronchographic changes were narrowing and poor

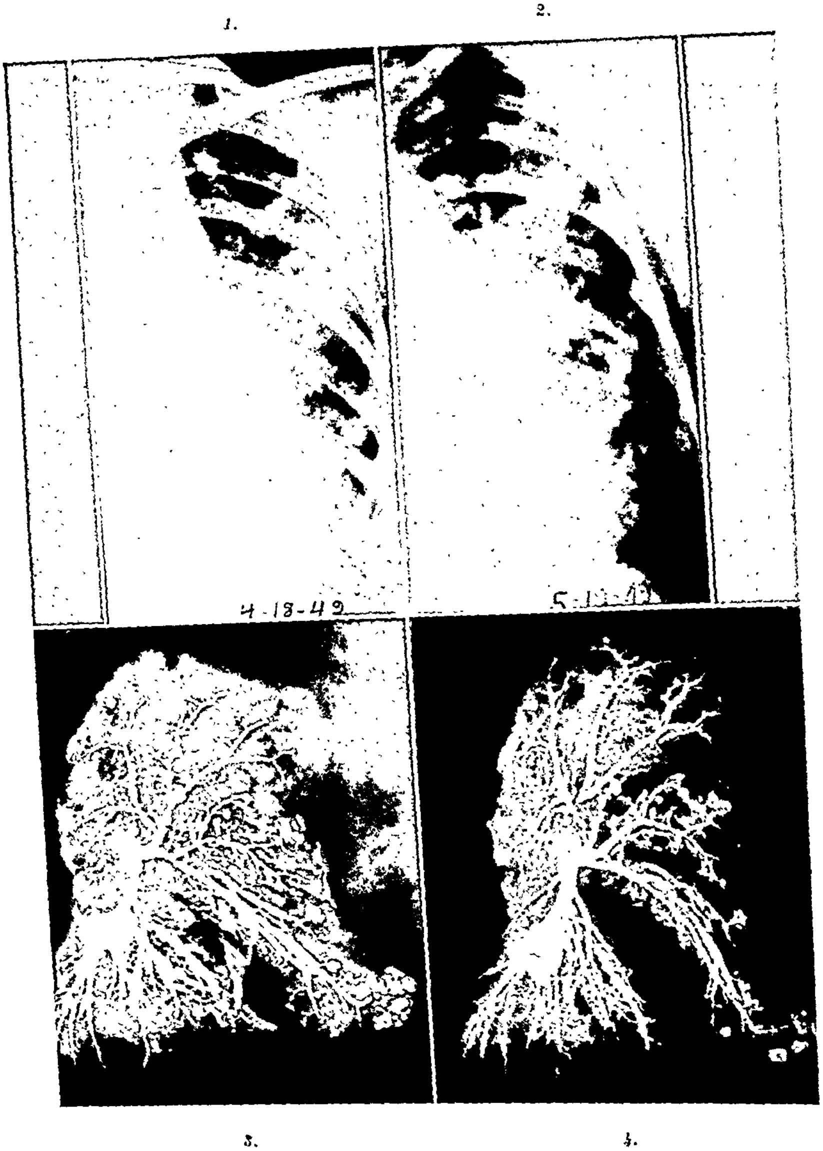


Fig. 3.—*1*, (*Case 6*) Posteroanterior roentgenogram, April 18, 1949, showing moderately extensive shadows on the left. *2*, (*Case 6*) Posteroanterior roentgenogram, May 19, 1949, showing new shadows of recent extension of disease. *3*, (*Case 6*) Roentgenogram after bronchial tree injection showing narrowing and lack of branching through diseased lung. At cavity which is filled the bronchus is short and wider than normal. *4*, (*Case 6*) Same specimen with addition of pulmonary arterial injection showing some relatively normal pattern in areas of recent disease and uninvolved parts of lung.

filling of some of the smaller bronchi (Cases 16, 18, and 19) so that normal arborization was lacking. With more extensive disease the distal portions of the bronchi did not fill at all, while the proximal larger bronchi were dilated (Cases 10, 11, 12, 13, 14, 15, 16, 19, 20, and 25), had irregular margins, and were

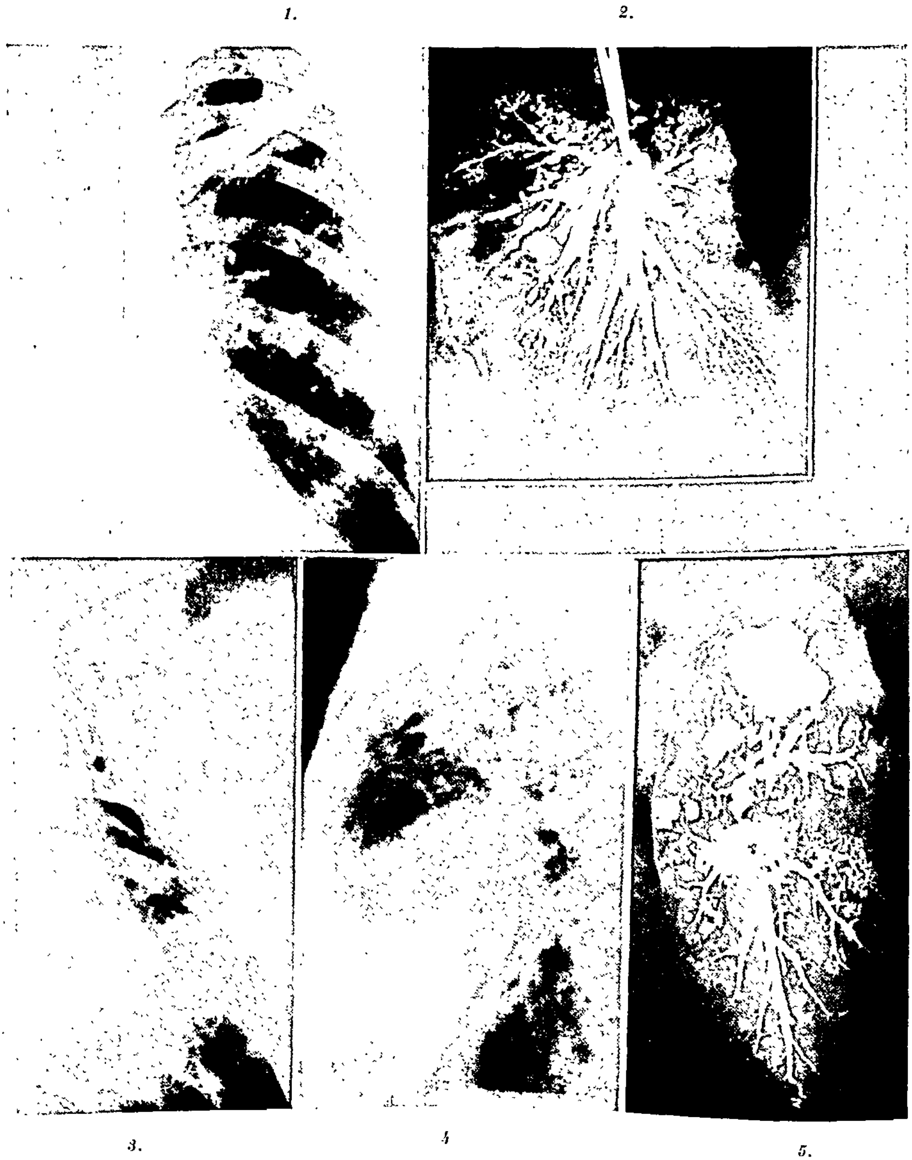


Fig. 4.—*1*, (*Case* 7) Posteroanterior roentgenogram of left lung showing few shadows. *2*, (*Case* 7) (Left pneumonectomy)—Roentgenogram after injection of both bronchial tree and pulmonary artery showing filling chiefly of larger divisions. (See pathologic description in Fig. 5.) *3*, (*Case* 8) Posteroanterior roentgenogram showing annular shadow—thoracoplasty failure—extension of disease into lower lung field. *4*, (*Case* 8) Left lateral roentgenogram showing location of disease in lingula. *5*, (*Case* 8) (Left upper lobectomy)—Roentgenogram after injection of bronchial tree showing only filling of cavity—bronchi to upper segment in region of cavity are short, wide, and have almost no branches, bronchi in lower segment showing narrowing and only few branches. (See pathologic description in Fig. 6.)

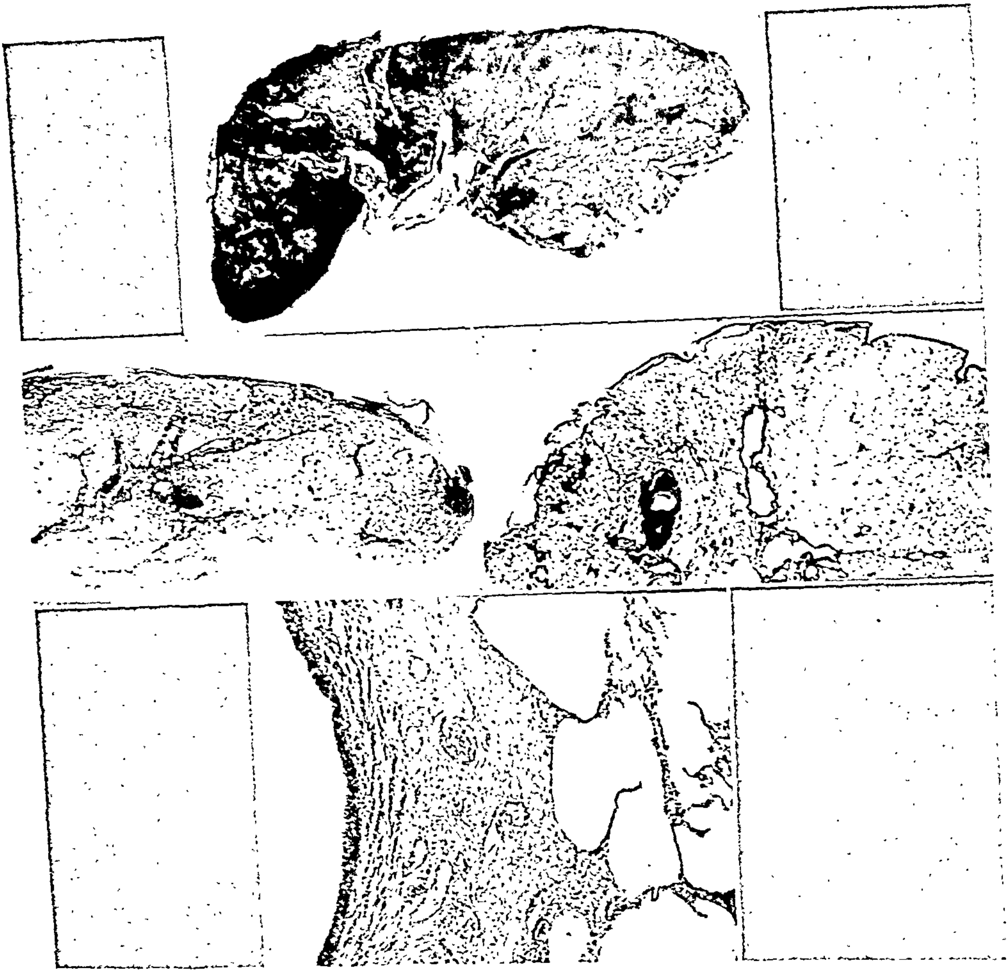


Fig. 5 (*Case 7*).—*1*, Gross photograph of left lung removed at operation. The upper lobe is at the left. Though rather few bronchi and vessels are seen, chiefly in cross section, it is apparent that all of them course through air containing pulmonary tissue. Note that the alveoli are generally coarse. The paucity of shadows in the chest roentgenograms is readily explainable by the appearance of the gross lung. The reasons for bronchographic changes, however, are less apparent. These changes depend upon the specific lesions associated with the bronchi, which are better shown in *2*, *3*, and *4*.

2, Low-power photograph of the histologic preparation from the lower lobe. Bronchi and vessels are seen extending into the lobe from the left. Their walls are thickened and their linings are irregular. The scattered minute round or elongated spaces with dark walls are bronchioles and arterioles which, though patent, were not outlined in the bronchograms. (Approximately ×1.5.)

3, Low-power photograph of the histologic preparation of the upper lobe of the lung shown in *1*. The oval dense focus is a dilated bronchus filled with exudate. At the center is a similarly dilated, but empty bronchus. At the left, beneath the pleura, are several small foci of encapsulated caseous material. In all areas there are minute patent bronchioles and arterioles seen in longitudinal and cross section. Generalized coarsening of the alveoli is apparent. (Approximately ×1.5.)

4, Photomicrograph of the wall of a bronchus which measured about 3 mm. in diameter in the section. The epithelium is intact, but the wall is greatly thickened by a dense concentric zone of scarring. The smooth muscle of the wall is obliterated. Dilatation and rupture (emphysema) of the adjacent alveoli is apparent. The contrast between this lesion and that shown in Case 8, Fig. 6, *4* is striking and apparently fundamental. (×70.)

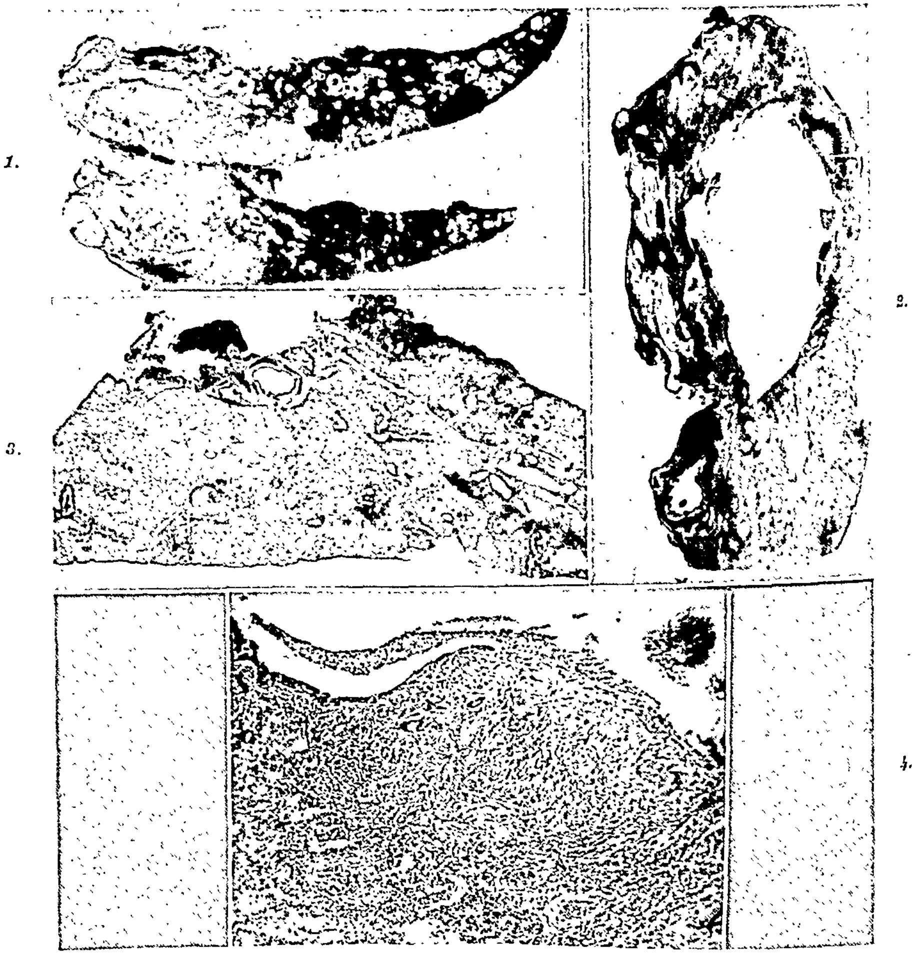


Fig. 6 (*Case 8*).— *1*, Gross photograph of two blocks of the left upper lobe removed at operation. The apex is at the left. The upper block shows a large cavity in the apical region. The lower block is more posterior and shows a rather extensive area of scar tissue and caseous material. The hilar bronchi show distinct thickening of their walls, and bronchi and vessels are largely obscured in the upper third of the lobe. In the lower portion of the lobe are seen numerous white foci of recently disseminated disease. Close inspection of these reveals that most of them are within or closely adjacent to small bronchi, the lumens of which can be identified. Close inspection also reveals many small vascular lumens, without associated foci of disease. These findings correlate with the roentgenogram studies which revealed poor filling of the bronchi, but good filling of the vascular tree. The dark areas are the result of hemorrhage into some of the secondary lobules during operation.

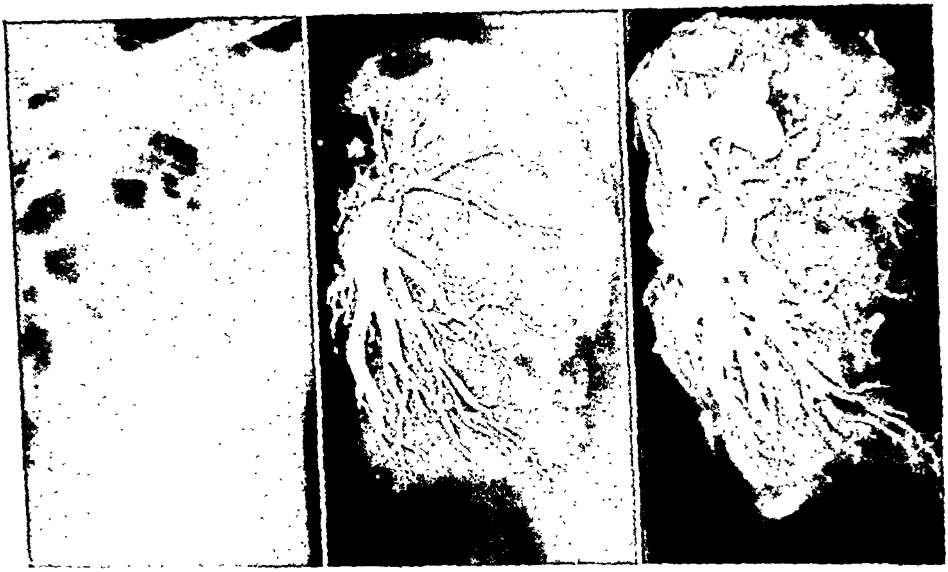
2, Low-power photograph of the histologic preparation from upper half of the lobe shown in *1*. In this section there are few bronchi, but the two adjacent to the lower border of the cavity show thickened walls and serrated lumens, like those in Case 7, (Fig. 5, *1*) (Approximately ×2.)

3, Low-power photograph of the histologic preparation from the lower half of the lobe shown in *1*. Operative hemorrhage obscures much of the field, and the lesions are best seen toward the left. Here several bronchi are seen in longitudinal section. Their walls are thickened and immediately adjacent alveolar tissue is consolidated. (Approximately ×2.)

4, Photomicrograph of the wall of a bronchus of similar size to that shown in Case 8, Fig. 5, *4*. The epithelium is ulcerated, but regenerating from the right, and a well-developed tubercle is protruding into the lumen. Adjacent alveoli are consolidated by exudate. It is likely that this is the early stage of the type of lesion which results finally in the peribronchial fibrosis seen in Case 7, Fig. 5, *4*.

crowded together.† The bronchograms in living patients did not appear to differ essentially from those of removed lung specimens, except that in the latter cavity filling was the rule rather than the exception.

Once these bronchial changes of chronic tuberculosis have occurred they appear to be irreversible, even though in some instances the diseased portion of the lung again becomes radiolucent as seen by routine chest roentgenograms. This has been observed, for example, in some cases of tuberculous pneumonia treated with streptomycin and P.A.S. Following the use of these drugs marked clearing of the dense shadows occurred leaving only linear strands. Bronchograms done one month to two years after this clearing showed an unanticipated extent of alteration in the bronchial pattern (Cases 17 and 20). In other instances this clearing has apparently been accompanied by marked resolution,



1. 2. 3.

Fig. 7 (*Case 9*).—*1,* Posteroanterior roentgenogram showing shadows of extensively destroyed left lung. Patient originally had cavity in lower lobe treated by pneumothorax. Ultimate re-expansion followed in few years by general breakdown of entire lung. *2,* (Left pneumonectomy)—Roentgenogram after injection of pulmonary arterial system indicating general paucity of pattern showing small lingular *segment with* some few branches, contracted lower lobe with similar absence of branching. *3,* Same specimen with addition of bronchial tree injection showing filled cavities and short wide bronchi with lack of branching. (See pathologic description specimen in Fig. 8.)

Case 9.—Pulmonary Function Study

Estimated vital capacity	5.02 L.
Determined vital capacity	3.85 L.
Estimated maximum breathing capacity	182 L.
Determined maximum breathing capacity	125 L.

By fluoroscopy the movement of the right diaphragm is within normal limits and the left diaphragm cannot be visualized. The right lung ventilates normally. The mediastinum is shifted moderately to the left on inspiration and returns toward the midline on expiration. There is little evidence of function on the left.

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†The mechanism responsible for the bronchographic patterns is not clear and is under further investigation. It is thought that the latter finding may be due to partial collapse and retraction resulting from destruction of intervening parenchyma. The dynamics of the lung are such that the bronchi enlarge and elongate during inspiration and shorten and narrow in expiration. The damaged portion of the lung takes little or no part in normal respiratory function because of marked structural change including loss of elastic tissue. In this relatively collapsed or poorly functioning state the larger bronchi become *fore-shortened* and widened.

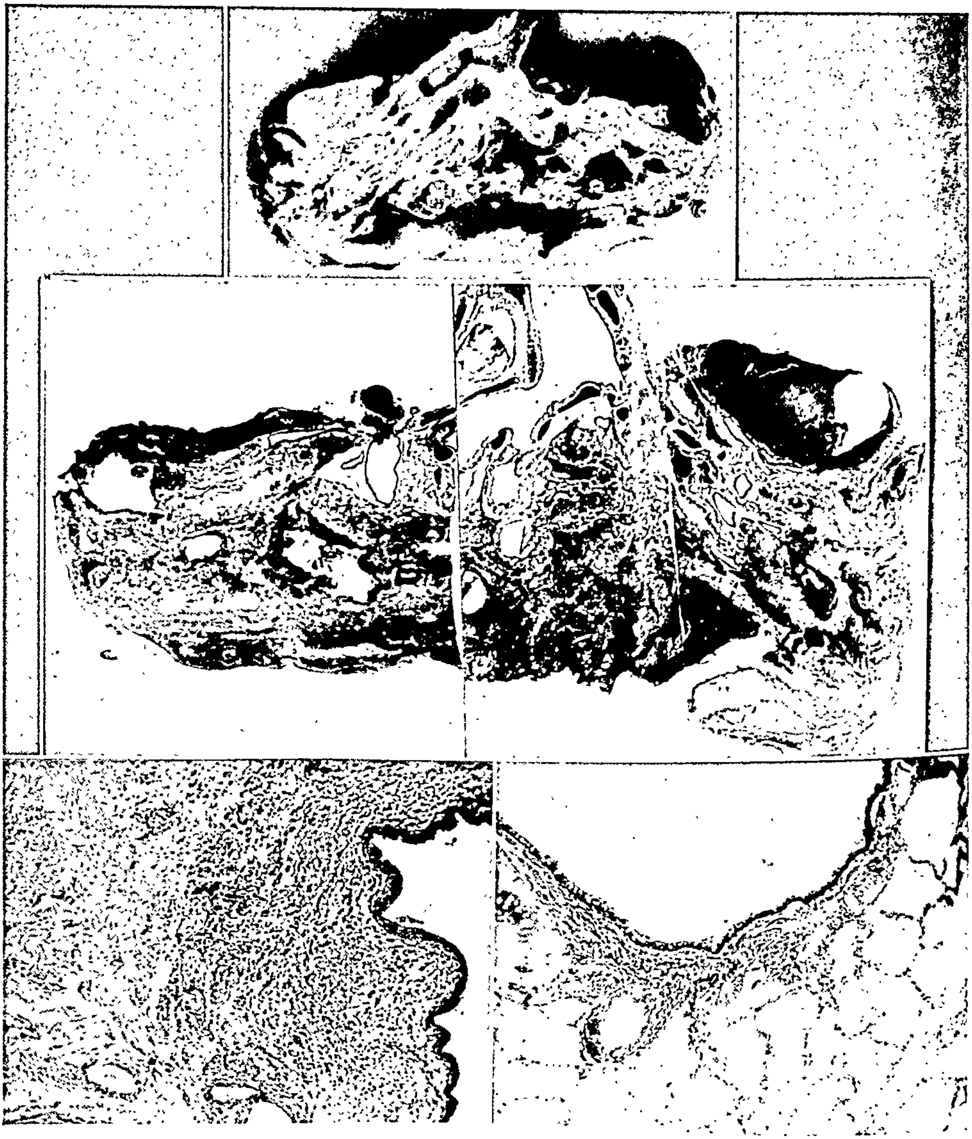


Fig. 8 (*Case 9*).—*1*, Gross photograph of the left lung removed at operation. The upper lobe is at the left, and the lower lobe is at the right. Two cavities are to be seen in the upper lobe, with a trabecula crossing the more lateral one. Between the two cavities a dilated tortuous bronchus is seen. It is this type of change, occurring in numerous bronchi, which produces the serrated appearance in the bronchograms. The same type of alteration is better seen in *2*, a composite photograph of the histologic preparations of this lung. The walls of the hilar bronchi are thickened, but their lumens are of essentially normal size. Hilar vessels, several of which may be seen between the main lobar bronchi, also show thickening of their walls, but essentially normal size. Smaller bronchi and vessels are effaced in the areas of scarring, or caseation which have almost completely replaced the alveolar structure throughout both lobes. *2*, Composite low-power photograph of the histologic preparations of the lung. The cavities noted in *1* are again seen in the upper lobe, and the virtually complete replacement of alveolar structure by scarring and caseation is apparent. The elongated structures with serrated linings are bronchi, chiefly of the second order. Many of these are dilated, and are embedded in areas of scarring. The more smoothly outlined spaces are vessels of the pulmonary system. Smaller branches of both the bronchi and the vessels are effaced in the scar tissue or caseous material. (Approximately ×1.5.) *3*, Photomicrograph of the wall of one of the bronchi showing dilatation and serration of the lining. Note that the mucosa is intact. There is moderate lymphocyte infiltration beneath the mucosa. At the left is shown a portion of the wide area of diffuse collagen deposition through which the bronchus courses. All evidences of bronchioles, alveolar ducts, and alveoli are effaced. Compare with Fig. 5, *4* which shows localized peribronchial scarring with preservation of adjacent alveolar structure. *4*, Photomicrograph of a normal bronchus of similar size to those in Figs. 4 and 8. Note the cartilagenous plate at the left. The epithelium is intact.

and the bronchial change seen was small in degree. On the other hand, the bronchographic pattern remained normal in areas where the parenchyma was not diseased but the lung collapsed from without, as in cases of pneumothorax (Cases 23 and 24), pleural effusion (Case 10), or thoracoplasty (Cases 17 and 21).

Bronchograms were also obtained in a number of patients who had pulmonary function studies (Cases 9, 13, 19, and 21) carried out preliminary to major chest surgery. It was hoped that some means of estimating function with the aid of bronchography might be worked out. To date insufficient material has been studied by both methods so that no conclusions can be

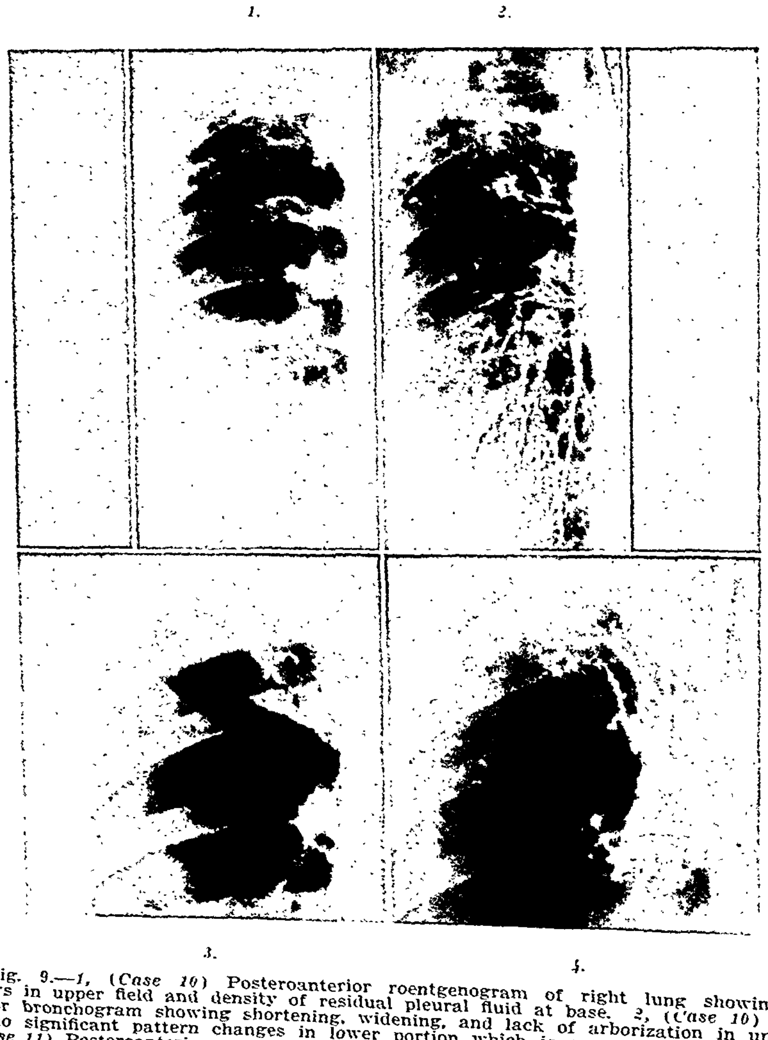


Fig. 9.—*1*, (*Case 10*) Posteroanterior roentgenogram of right lung showing linear shadows in upper field and density of residual pleural fluid at base. *2*, (*Case 10*) Posteroanterior bronchogram showing shortening, widening, and lack of arborization in upper lobe with no significant pattern changes in lower portion which is compressed by pleural fluid. *3*, (*Case 11*) Posteroanterior roentgenogram showing density at apex. Question as to whether this represents contracted segment or lobe. *4*, (*Case 11*) Bronchogram showing shadow representing entire right upper lobe contracted.

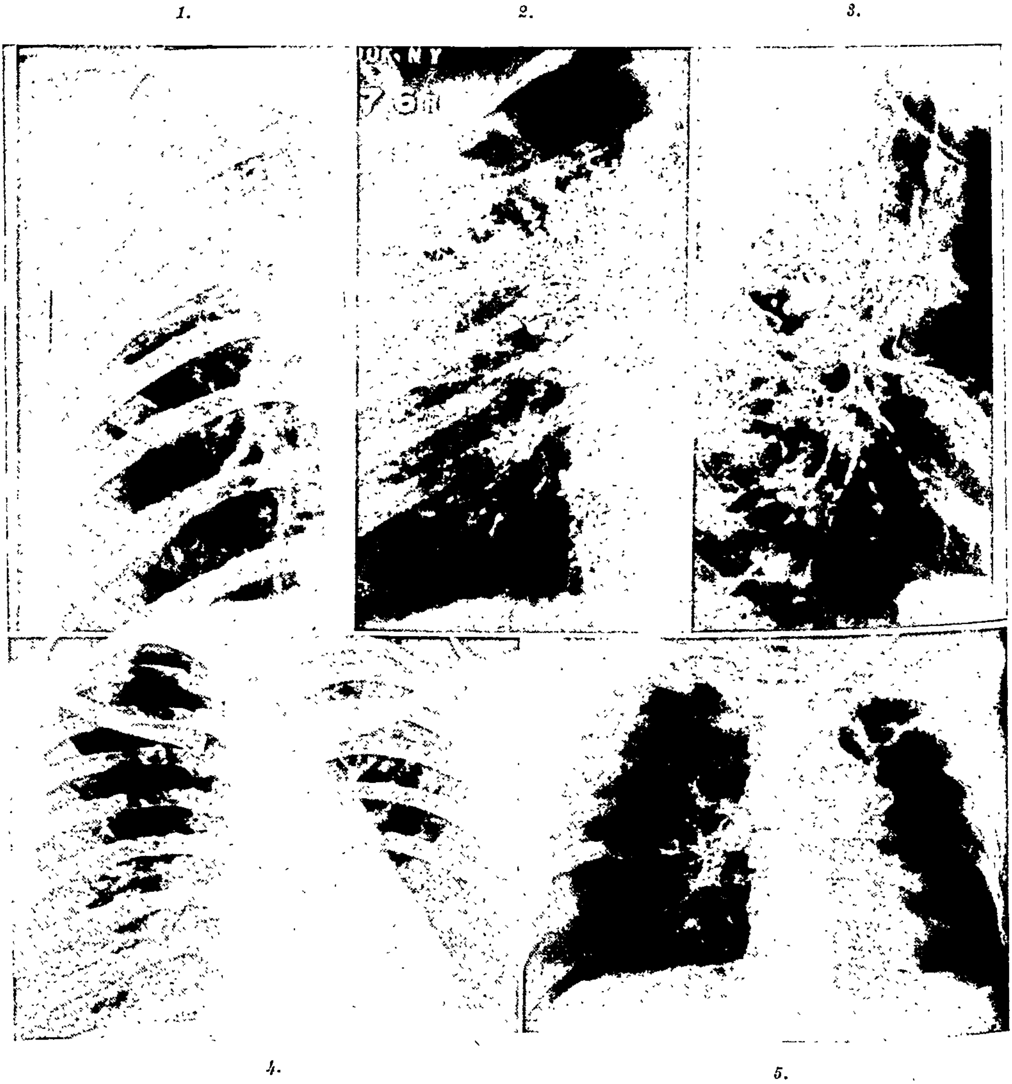


Fig. 10.—*1,* (*Case 12*) Posteroanterior roentgenogram showing extensive abnormal shadows in upper lung field. *2* and *3,* Posteroanterior and right lateral bronchogram showing characteristic short, wide bronchi in most of upper lobe and apex of lower lobe. *4,* (*Case 13*) Posteroanterior roentgenogram showing small annular shadow in upper lung field, left and mediastinal displacement. *5,* (*Case 13*) Bronchogram showing very poor filling throughout most of the left lung except at base despite every effort to improve this by addition of contrast medium. This latter only resulted in filling of trachea, spill to opposite lung, and ingestion into esophagus.

Case 13.—Pulmonary Function Study

Estimated normal vital capacity	3.6 L.
Determined vital capacity	1.9 L.
Determined resting minute ventilation	9.2 L.
Determined maximum minute ventilation	68.0 L.

The right diaphragm moves normally, but the left has only slight movement. There is good clouding on the right in expiration, poor on the left and the mediastinum shifts to the right slightly. By fluoroscopy it is estimated that the right lung is doing 75 per cent of the ventilation.

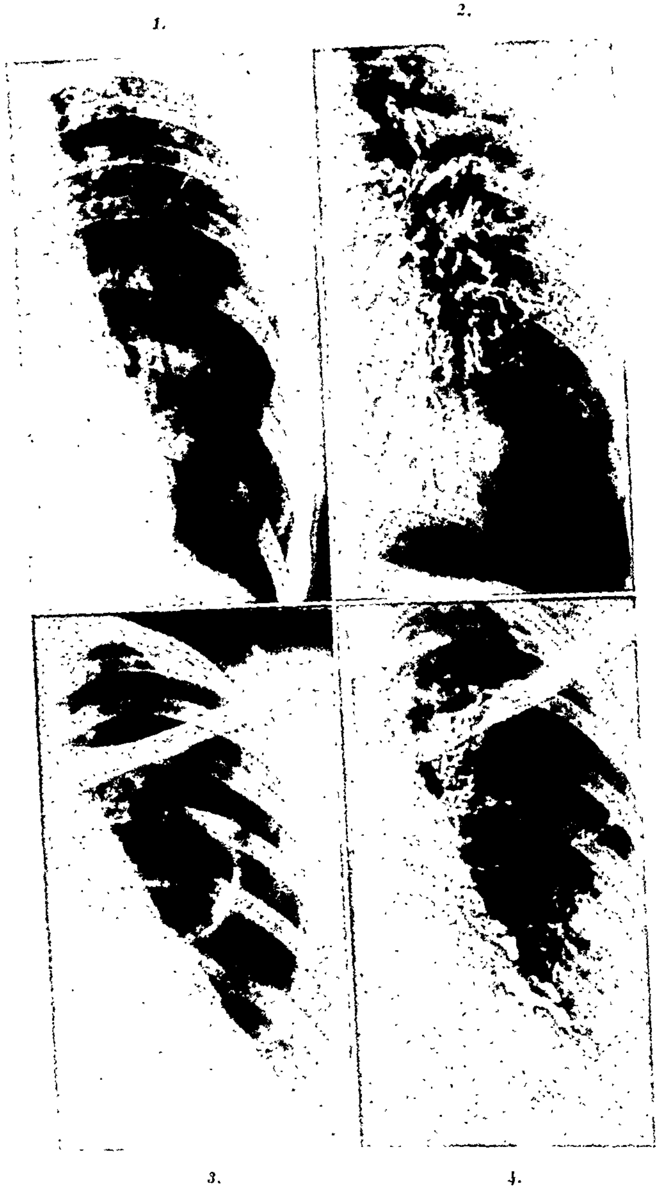
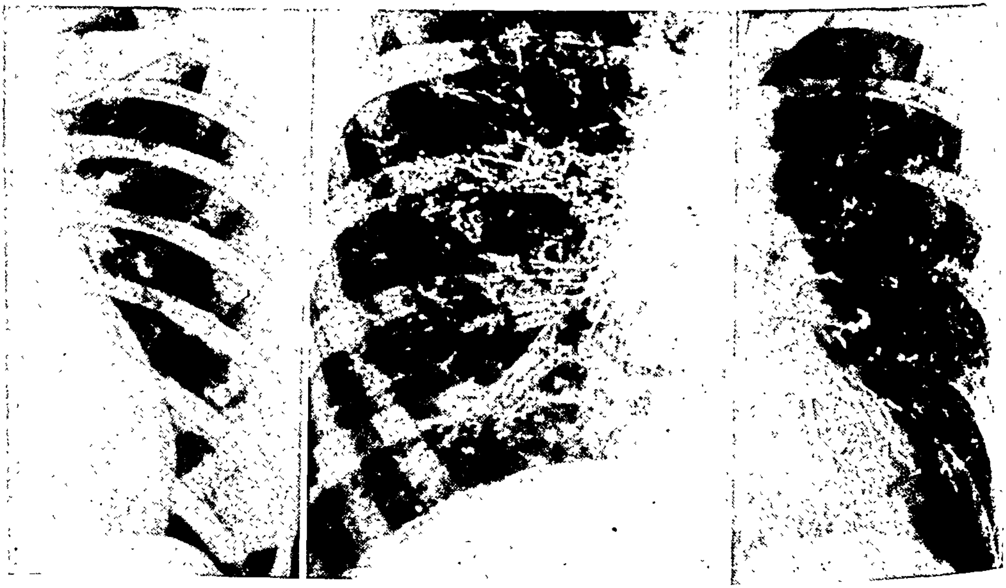


Fig. 11.—*1*, (*Case 14*) Posteroanterior roentgenogram showing few scattered abnormal shadows in upper lung field and retracted root shadow. *2*, (*Case 14*) Posteroanterior bronchogram showing marked changes through most of the lung and revealing associated bronchial defects including emphysema. *3*, (*Case 15*) Posteroanterior roentgenogram showing few linear shadows at apex and giant-sized annular shadow identified as being in the lower lobe on lateral film. *4*, (*Case 15*) Posteroanterior bronchogram showing characteristic abnormal pattern in upper lobe and marked changes through the lower lobe. Only the lingular segment shows a normal pattern.

normal anatomy. That is, one cannot tell to what extent the previously diseased lung has been damaged, even though the tissue contains air and is therefore radiolucent. In inexpandible lungs, as sometimes occurs after pneumothorax, the routine chest films give very little information regarding the condition of the collapsed lung, while bronchograms still distinguish diseased areas in such cases in the collapsed as in the uncollapsed lung (Cases 22, 23, and 25). Planigraphic studies of the chest are distinctly more valuable than regular chest films, but at open chest operation it is not uncommon to find more disease than seen in the films and at times areas of disease not seen.



1. *2.* *3.*

Fig. 14 (Case 18).—*1,* Posteroanterior roentgenogram showing few shadows in left upper lung field. The right side was normal. *2* and *3,* Posteroanterior bronchograms showing right side, especially of lower lobe region which has a normal pattern in contrast to thin narrow tree with fewer branches at left base. At operation the lower lobe was found studded with discrete tuberculous nodules along the course of the bronchi and the involved segments of lung were excised. (Compare with Case 8, Fig. 4.)

Bronchoscopy gives valuable information in certain cases. However, its value is limited to the larger bronchi. Bronchography, in contrast, reveals the changes beyond the visual field of the scope.

The question of possible harmful effects is frequently raised when iodized oil is suggested for studying cases of pulmonary tuberculosis. The material has been used here for years in patients with both positive and negative sputum. A careful study of postbronchographic films over long periods has failed to show any harmful effects attributable to the iodized oil. When the examination is made with care and minimum quantities of oil are used, practically all of this is removed when the patient utilizes postural drainage after films are taken.

tensively diseased or collapsed lobe or lung is much smaller in size, ng requires only a small amount of material. If continued ill larger quantities of oil than needed, flooding of other areas of the lung will result. Retention of some of the oil will then be noted for weeks or months, and may make the roentgenographic follow-up troublesome in interpreting spreads of disease. However, a careful study of films taken before and shortly after bronchography largely solves this problem.

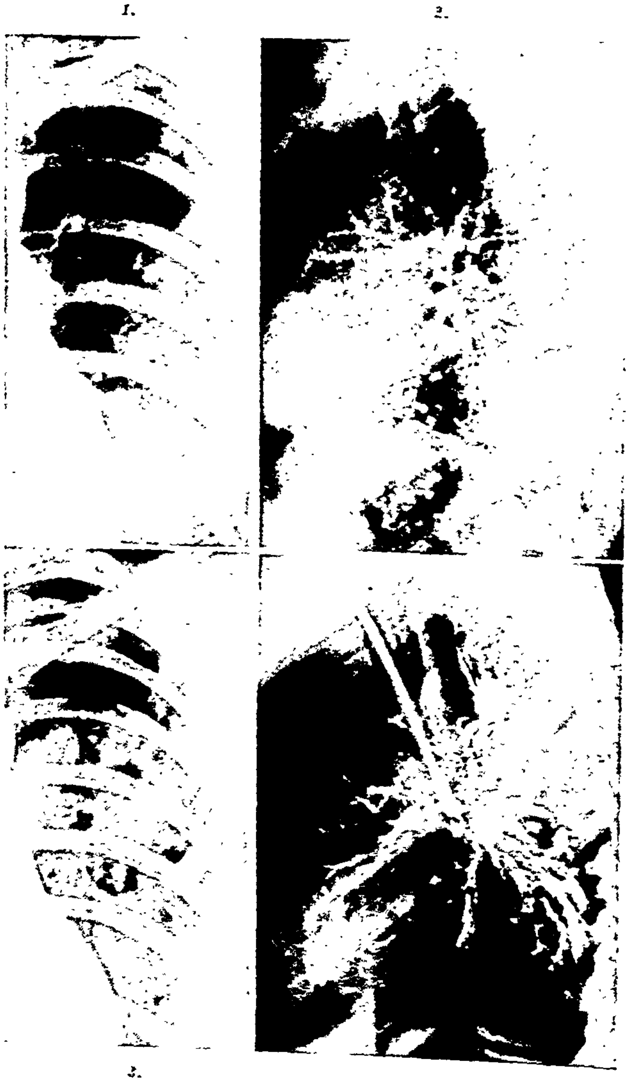


Fig. 15 (*Case 19*).—*1*, Posteroanterior roentgenogram showing abnormal density in lower lung region. *2*, Lateral bronchogram showing marked contraction of entire lower lobe with short, wide bronchi and few branches—the upper lobe shows some distension but a relatively normal bronchial pattern. *3*, Posteroanterior roentgenogram showing new shadows in mid lung field. *4*, Lateral bronchogram showing alteration of bronchial tree in region of new disease which previously showed normal pattern.

Case 19.—Pulmonary Function Study

Estimated normal vital capacity	3.25 L.
Determined vital capacity	3.15 L.
Determined resting minute ventilation	11 L.
Maximum minute ventilation	92 L.

By fluoroscopy the function on the right is normal, and the left lung functions fairly well except the lower lobe.

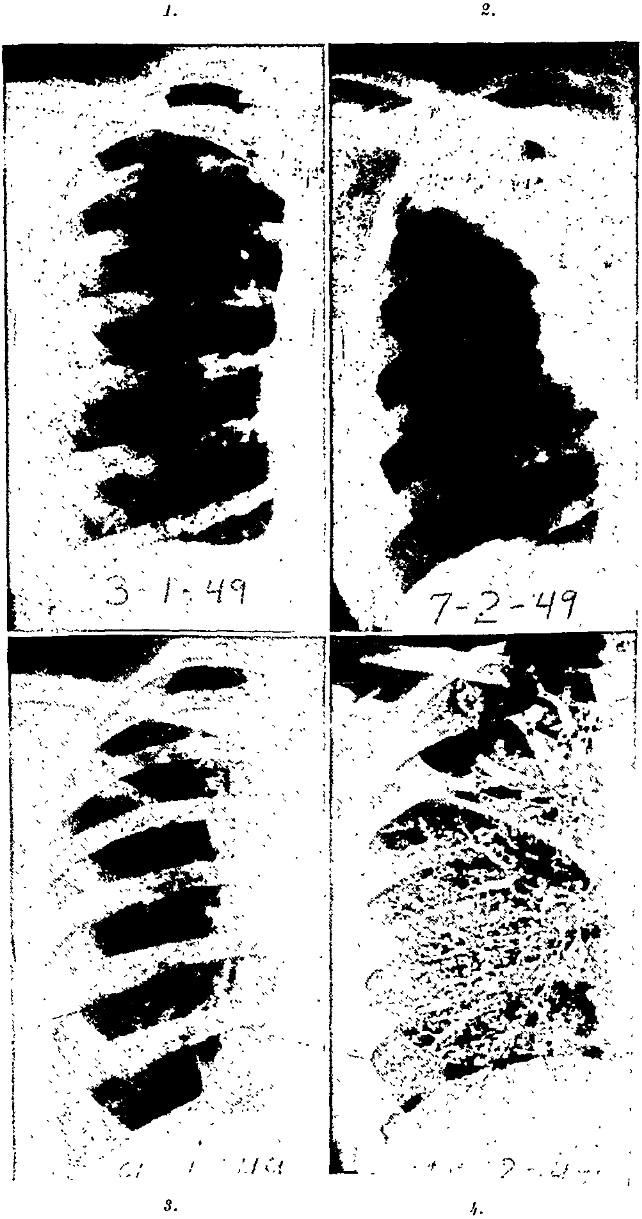


Fig. 16 (*Case 20*).—*1*, Posteroanterior roentgenogram, March 1, 1949, of patient with known minimal disease in extreme apex. *2*, Posteroanterior roentgenogram, July 2, 1949, after recent onset of tuberculous pneumonia showing dense wedge shaped shadow. *3*, Posteroanterior roentgenogram, Sept. 1, 1949, showing marked clearing of shadows with aeration of upper lung region and few linear strands. Film taken after two months treatment with streptomycin and PAS. *4*, Posteroanterior bronchogram, Sept. 22, 1949, showing marked changes in bronchial pattern of upper lobe. The rest of the tree appears normal.

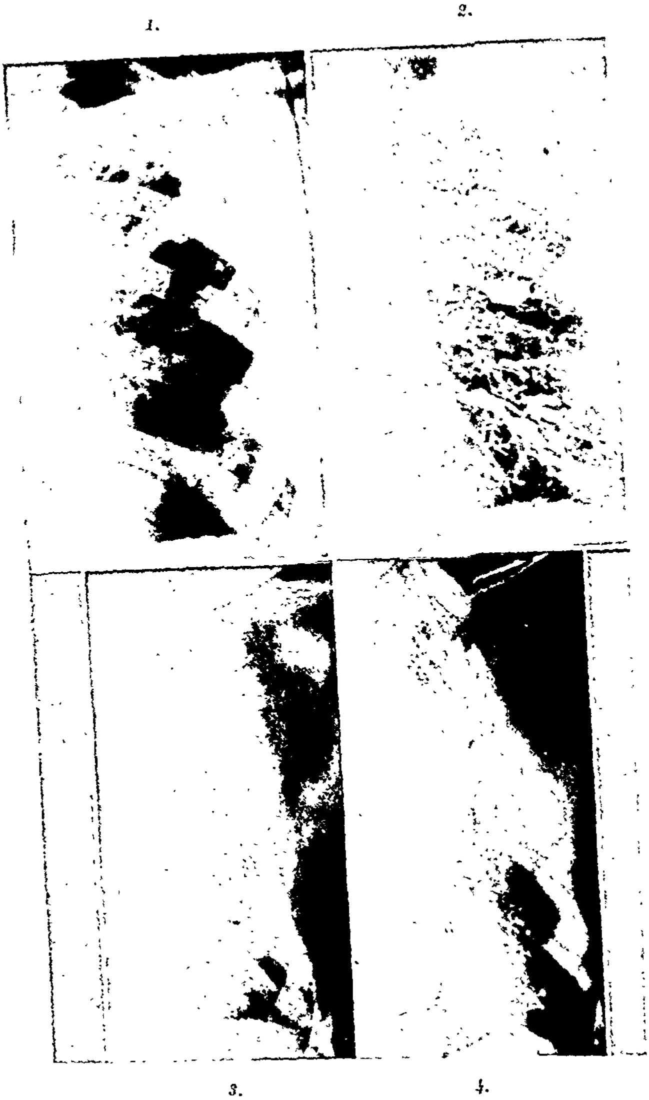


Fig. 17 (*Case 21*).—*1*, Posteroanterior roentgenogram showing annular shadows in upper lung field and at base. *2*, Posteroanterior bronchogram showing shadows of cystic abnormalities as well as alteration of bronchi. *3*, Posteroanterior roentgenogram—postthoracoplasty. *4*, Posteroanterior bronchogram showing persistence of abnormal shadows of bronchial tree after thoracoplasty.

Case 21.—Pulmonary Function Study Before Thoracoplasty

Estimated normal vital capacity	4.8 L.
Determined vital capacity	3.05 L.
Determined resting minute ventilation	16 L.
Determined maximum minute ventilation	120 L.

The diaphragms move well on both sides and the clouding during expiration is good on the right, but fair to poor on the left. The mediastinum shifts to the right on expiration. The greatest part of ventilation is being done by the right lung as studied by fluoroscopy.

Ten Months Postthoracoplasty

Estimated normal vital capacity	5.25 L.
Determined vital capacity	4.9 L.
Determined resting minute ventilation	9 L.
Determined maximum minute ventilation	92 L.

The diaphragm on the right moves well and there is good clouding on the right except at the apex. The diaphragm on the left moves poorly and the mediastinum shifts to the right on expiration. There is almost no ventilation on the left as studied by fluoroscopy.

After a few trial cases one can readily develop the necessary skill to be satisfied that the absence or poor filling of the bronchi is not due to technical fault. The abnormal bronchographic pattern in extensive parenchymal involvement so well depicted in Cases 12, 17, and 20 and in discrete dissemination in Cases 16, 18, and 19 need not be confused with incomplete filling of normal bronchi which would show normal caliber and some normal arboriza-

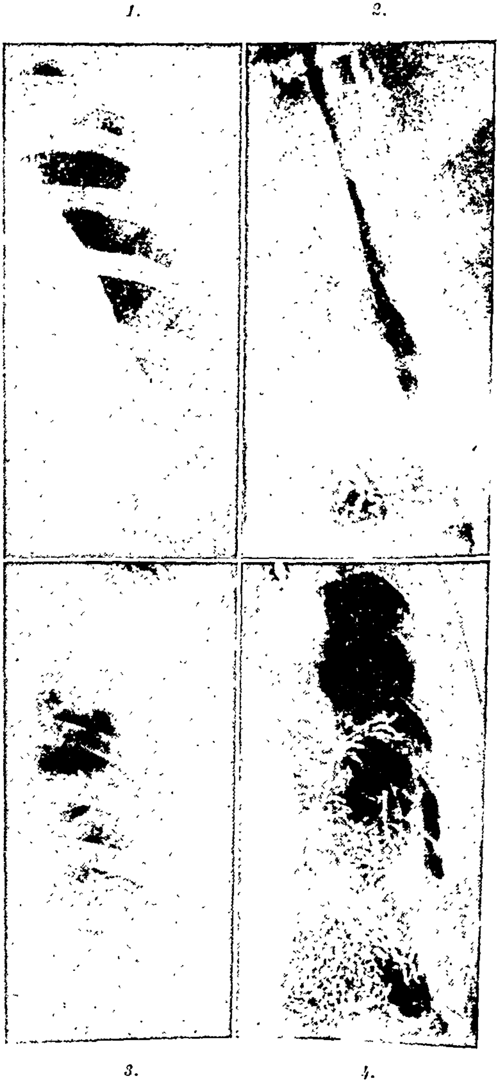


Fig. 18.—*1*, (*Case 22*) Posteroanterior roentgenogram showing totally collapsed lung proved to be inexpandible. *2*, (*Case 22*) Posteroanterior roentgenogram postthoracoplasty showing residual space and bronchogram showing poor pattern generally. Two attempts at reexpansion by decortication failed. *3*, (*Case 23*) Posteroanterior roentgenogram showing residual pleural space at base in case of oleothorax and a known bronchopleural fistula and empyema. *4*, (*Case 23*) Posteroanterior bronchogram showing normal pattern in lower lobe and shadow of catheter draining pleural space. Decortication and repair of fistula resulted in obliteration of pleural space by expansion.

tion. Patients with actively progressive disease or excessive secretions should not be chosen for this examination because of risk of spread of disease or retention of secretions when the cough reflex is temporarily blocked by topical anesthesia (0.5 per cent pontocaine solution).

It is helpful to have bronchoscoped the patient sometime before to ascertain the condition of the bronchial tree, noting any obstructive lesions and the disposition of the orifices to the lobes. This serves as a guide in positioning

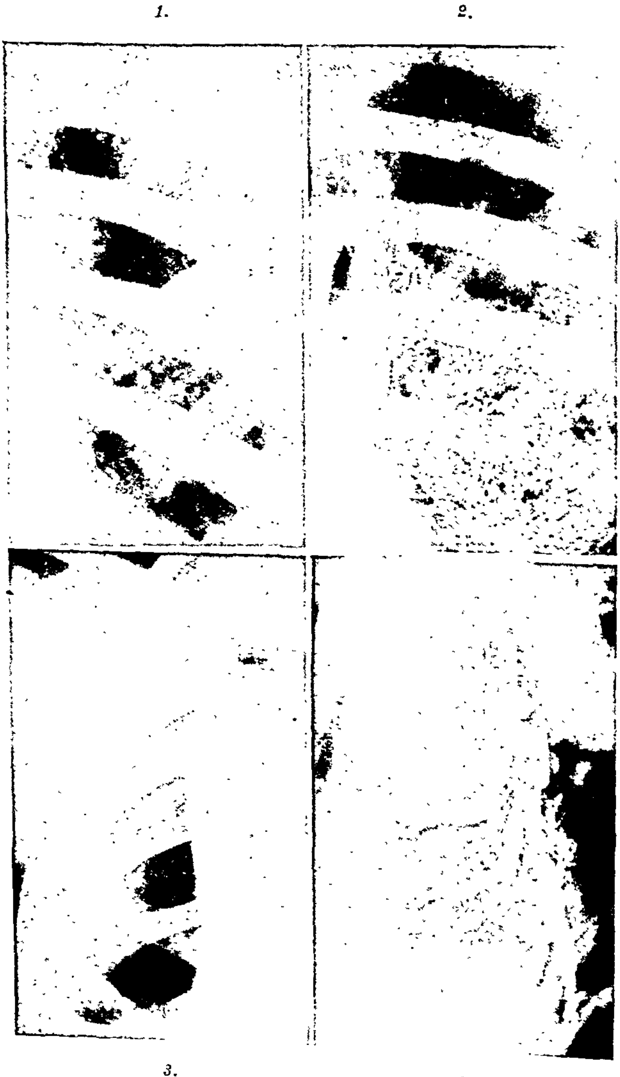


Fig. 19.—*1*, (*Case 24*) Posteroanterior roentgenogram showing annular shadow in upper lung field. *2*, (*Case 24*) Posteroanterior bronchogram showing abnormal bronchi in the involved part of lung only and no change elsewhere despite presence of pneumothorax. *3*, (*Case 25*) Posteroanterior roentgenogram of unexpandible lung in presence of pyopneumothorax. *4*, (*Case 25*) Oblique bronchogram showing marked alteration of bronchi to all lobes, indicating that reexpansion may not be anticipated with decortication.

the patient during the bronchographic examination. The combination of bronchoscopy and instillation of iodized oil is not advised, as it has not met with the success of doing the latter as a separate procedure at another time. An exception to this is the patient with a high degree of stenosis of a main

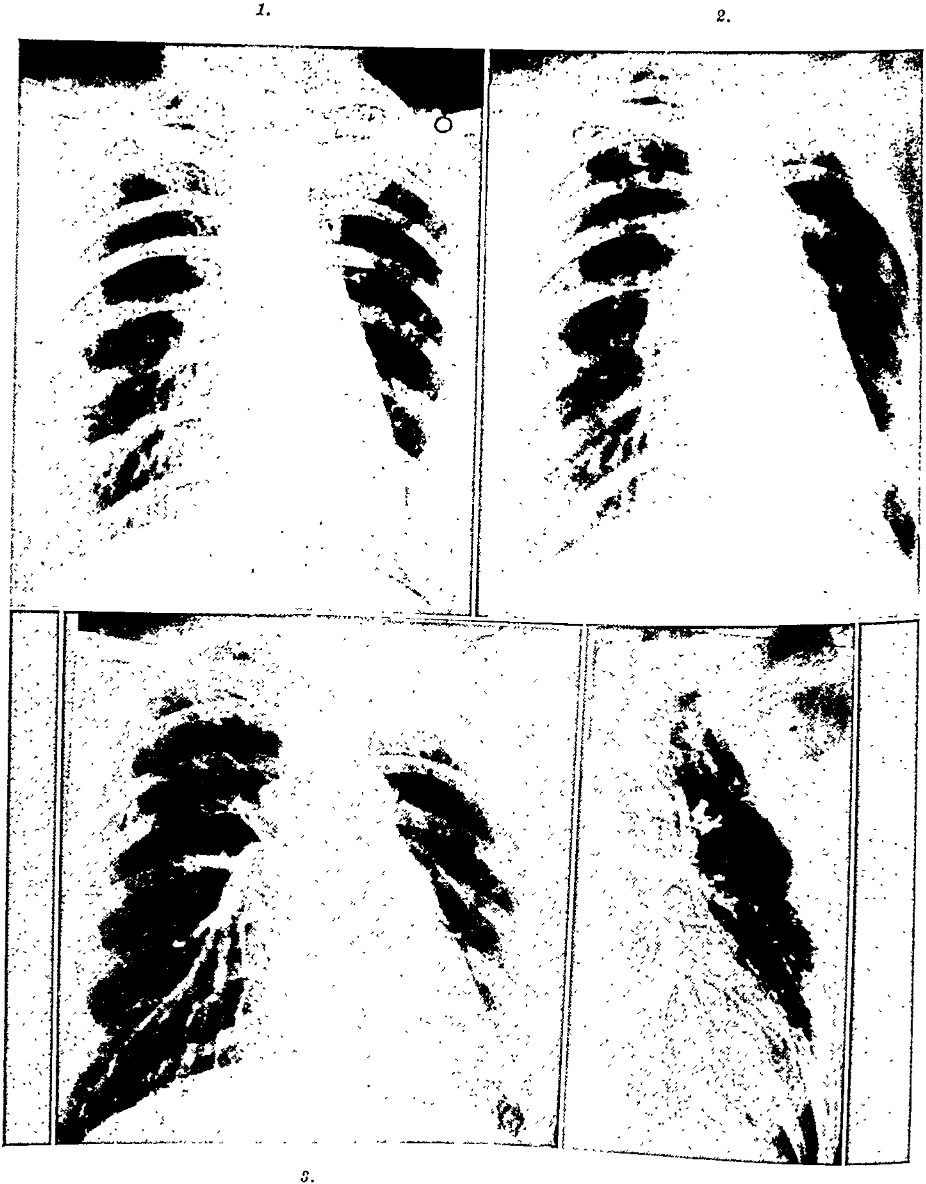


Fig. 20 (*Case 26*).—*1*, Posteroanterior roentgenogram showing small annular shadow left upper. *2*, Posteroanterior roentgenogram following first stage thoracoplasty. *3*, Posteroanterior angiocardiogram showing decrease in pulmonary arterial pattern in left lung and right upper which is site of old disease. *4*, Posteroanterior bronchogram of left showing decrease in arborization left upper and superior segment of lower. At operation disease involved much of apical-posterior segment of upper and superior segment of lower which were excised.

bronchus. Here the bronchoscopy is done on the roentgenogram table and, with the roentgenologist present, a watery contrast medium is instilled under fluoroscopic control. "Spot films" are taken followed by the immediate aspiration of the material and secretions. The use of nonoily solutions for

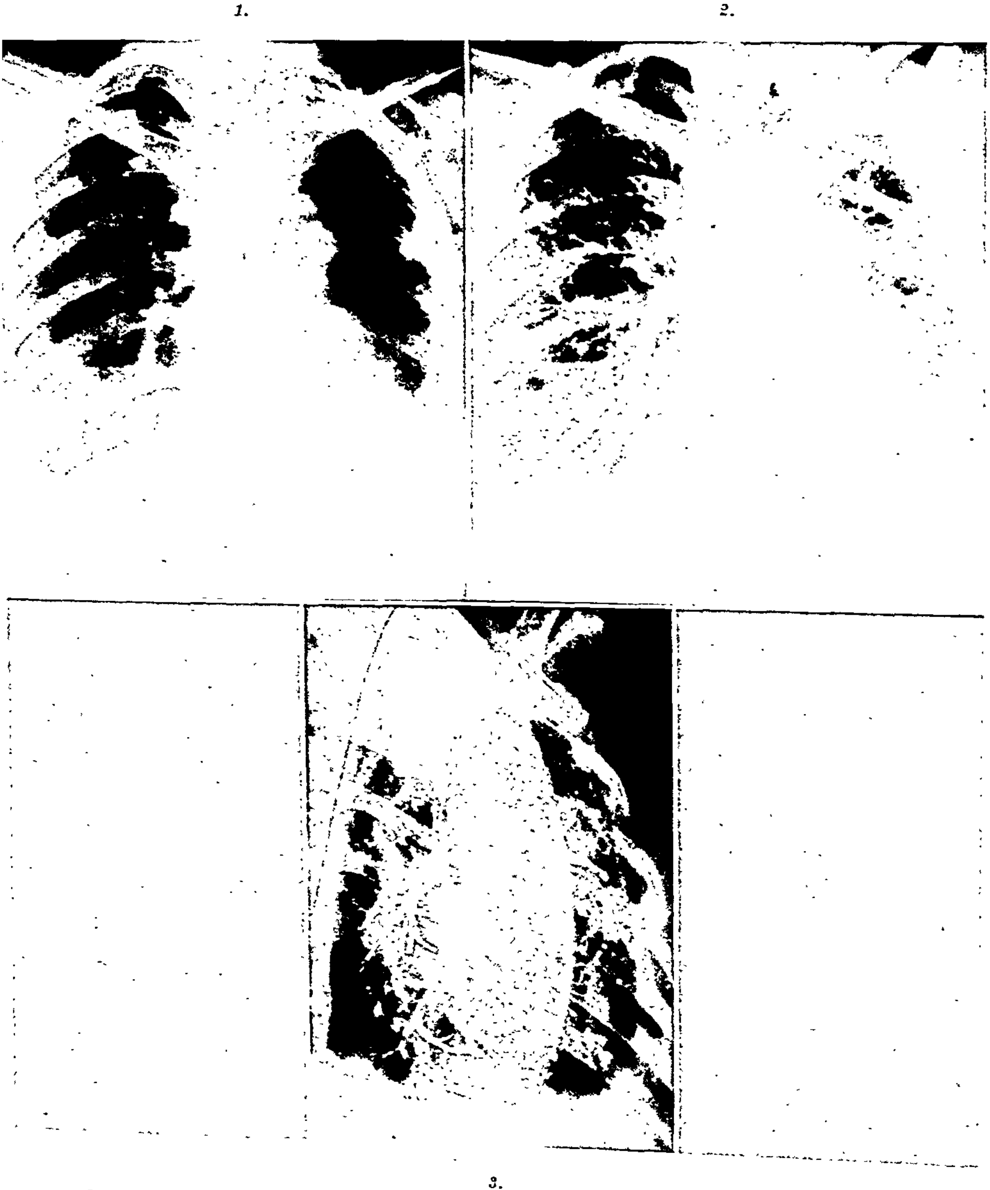


Fig. 21 (*Case 27*).—*1*, Posteroanterior roentgenogram in case of reexpanded (?) left pneumothorax showing mediastinal displacement cortical peel and linear density at site of previous cavity. *2*, Posteroanterior angiocardiogram showing fairly good pattern of left pulmonary artery except at linear density. Pulmonary arterial pattern on right is normal. *3*, Oblique bronchogram showing good pattern in all lobes except in same region as linear density where pulmonary arterial pattern was also decreased.

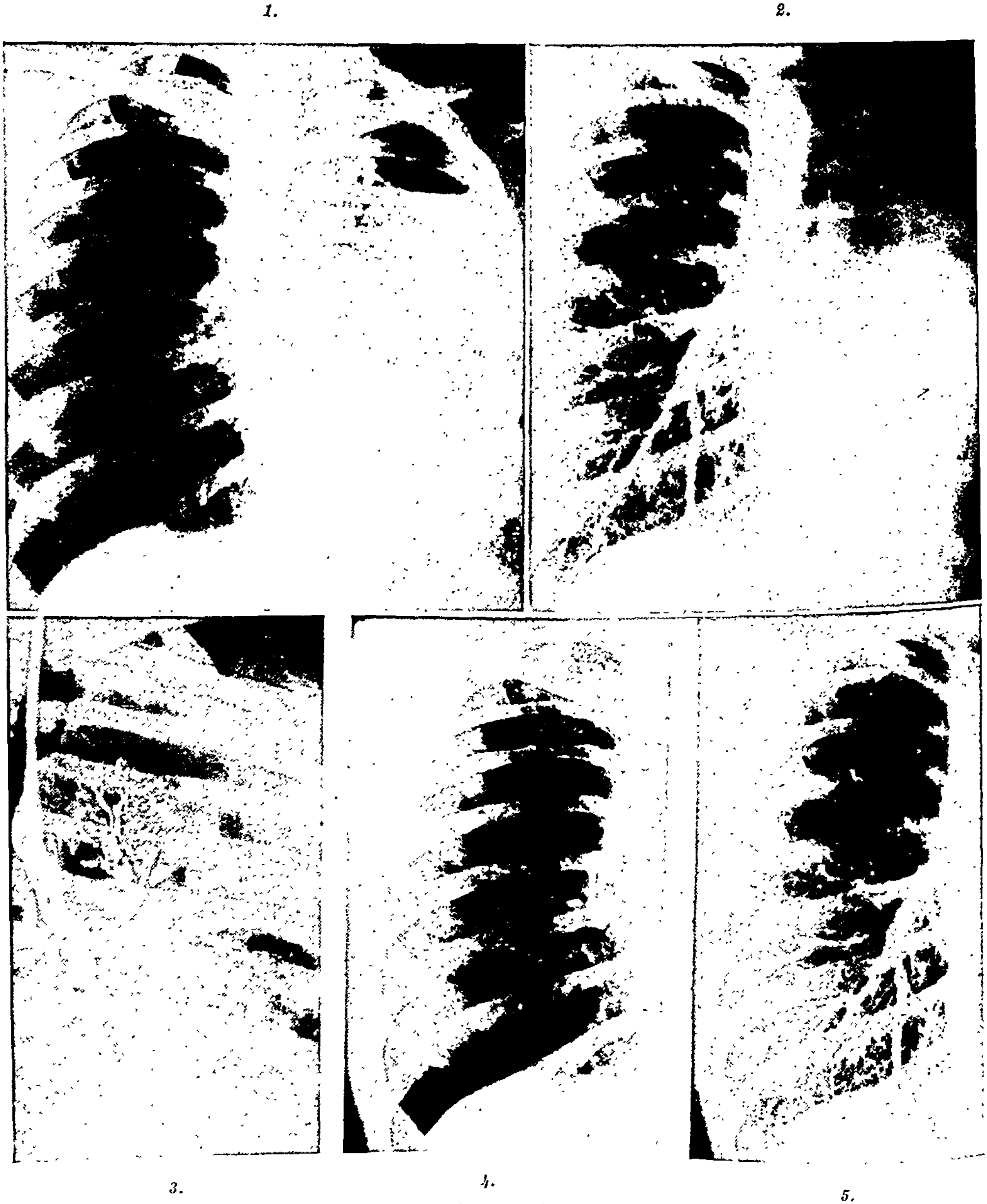


Fig. 22 (*Case* 28).—*1*, Posteroanterior roentgenogram showing unexpandible left lung with pyopneumothorax. *2*, Posteroanterior angiocardiogram showing absence of pattern of left pulmonary artery. *3*, Posteroanterior bronchogram left showing very poor bronchial pattern in all segments. *4*, Posteroanterior roentgenogram of right showing nodular densities in left upper lung field. *5*, Posteroanterior angiogram right side showing poor pulmonary arterial pattern in same area in contrast to normal pattern in middle and lower lobes.

routine bronchograms is not recommended, as it has not shown sufficiently satisfactory detail in the roentgenograms to be generally employed and is only used in the above situation. It is felt that bronchography gives such important information that certain disadvantages are outweighed. The examination can be a distinct aid in planning surgical treatment for pulmonary tuberculosis. The following are cited as illustrative examples of how bronchography can be helpful.

1. When resection of lung tissue is contemplated bronchograms aid in accurate localization of disease and thus help in deciding exactly which lobes or/and segments of lobes may need to be removed.

2. When thoracoplasty is considered it aids in helping to decide how extensive a procedure should be performed. Bronchograms frequently show evidence of marked structural changes, undoubtedly associated with damaged lung tissue, in areas where "clearing" has taken place as interpreted by routine chest roentgenograms. It is believed that such cases might fare better under permanent collapse (exemplified by Case 17). Perhaps prethoracoplasty bronchograms might also give a clue as to why some thoracoplasties are successful while others are not.

3. When decortication is considered to allow a "non-reexpansible" lung under pneumothorax to expand once more, bronchography gives the best information available as to the extent of structural changes present in the collapsed lung and thus aids in deciding whether the lung is healthy enough to make decortication advisable. (Exemplified by Cases 22, 23, and 25).

A PRELIMINARY NOTE ON PULMONARY ANGIOGRAPHY

Recently, with the aid of Dr. Charles T. Dotter, Radiologist at the New York Hospital, New York City, the technique of angiocardiography has been added. Dr. Berkeley Zinn, Supervising Radiologist at the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y., undertook a period of training under Dr. Dotter and returned here to add this method of study to the radiographic armamentarium. A study is under way to compare the pulmonary angiograms with bronchograms. Where possible injection study of suitable specimens as outlined in the early part of this paper will then be made before pathologic examinations are carried out and all material then correlated. Thus far, in a small series of cases, the anticipated correlation on the basis of injection studies of bronchi and pulmonary arteries has been observed. In Case 26 abnormal shadows of pulmonary tuberculosis were seen in both apices. A thoracoplasty was planned to collapse the small cavity in the summit of the left lung where an annular shadow was noted. After a one-stage, three-rib thoracoplasty was done an angiogram and bronchogram were performed at different times which indicated in both more structural alteration of the lung than suspected. On this basis pulmonary resection was chosen as the procedure of choice, and at operation the involved parts of the left upper and superior segment of the lower lobes were removed. Cases 27 and 28 were studied preliminary to consideration of decortication. Because of such good bronchial and pulmonary arterial patterns in the former decortication with limited resection of the involved portion of the lung was chosen in Case 27. In Case 28, because of widespread change in the bronchogram and very poor filling of the left pulmonary artery, pleuropneumonectomy was considered the procedure of choice.

SUMMARY

Tuberculous lung specimens, which were removed at operation or autopsy, were studied by injecting the bronchi and pulmonary arteries with radiopaque material. Pathologic studies of these lungs were made showing the extent of disease present. This study served as a basis for the interpretation of bronchograms in the living. It is believed that bronchograms give more information than is generally apparent as to the exact location and extent of disease present. It may also suggest the amount of functionally available lung tissue still present. Recently, angiocardiographic studies have been added in addition to bronchograms, and appear to correlate well with the injected lung studies that both systems are implicated in the structural alterations of the lung by chronic pulmonary tuberculosis. This information has been used in planning such types of surgical treatment of pulmonary tuberculosis as resection, thoracoplasty, and decortication. Numerous illustrations are included.

A note of thanks is made to the many physicians and surgeons in the Saranac Lake area for their excellent cooperation in permitting the use of surgical and postmortem specimens for study. Thanks are also extended to the resident staff members and technicians who cooperated in this work.

The radiographic illustrations were prepared by Anna M. Sanlow, Senior X-ray Technician, Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.

The pathologic illustrations were prepared by Edward Gockeler, Photographer, Saranac Laboratory, Saranac Lake, N. Y.

EFFECT OF POSITION AND ARTIFICAL VENTILATION ON THE EXCRETION OF CARBON DIOXIDE DURING THORACIC SURGERY

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OUR finding[5] that a dangerously high tension of carbon dioxide is common in the blood during open thoracic surgery has been widely confirmed.[6-9] It is now generally agreed that the tension of carbon dioxide will rise seriously during surgery with the pleura open unless active measures are taken to prevent it. We are not all agreed, however, as to the reason for this accumulation of carbon dioxide, nor are we all agreed as to the best method to get rid of the excess carbon dioxide. The purpose of this study was to try to answer the questions of why the carbon dioxide tension rises and of how to prevent this rise.

Elimination of carbon dioxide has been studied during thoracic surgery in the prone position used by Overholt and in the supine position, as well as in the lateral position with and without artificial assistance of ventilation. We showed in our original paper[5] that the pneumonectomy cases present the greatest problem. Therefore we have focused our principal attention on this group. A comparison of the data obtained when the patients are in the several positions demonstrates that position of the patient plays the major role in the development of or avoidance of a respiratory acidosis, in the absence of artificial respiration. Several other factors add to the patient's respiratory embarrassment: the existing pulmonary disease, the anesthesia, the open pleura, the crippling imposed during surgery; but these are, of course, present in all positions and cancel out in the comparisons.

Comparison of the work of the several groups now interested in this field permits some tentative conclusions as to the relative effectiveness of the two principal methods of artificial ventilation of the lungs, the hand and the machine.

We have shown already that it is easy to maintain full oxygenation in nearly all cases when patients are breathing the 96 volumes per cent oxygen possible with ether. Carbon dioxide excretion is the great problem; it is the subject of this paper.

METHODS

Conduct of Anesthesia.—Premedication in most instances consisted of pentobarbital sodium 100 or 200 mg., morphine 10 or 15 mg., and atropine 0.6 mg.

From the Anesthesia Laboratory of the Harvard Medical School at the Massachusetts General Hospital.

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The subjects to be operated on in the prone position received the higher dosages mentioned, and this can be assumed to account for the somewhat higher preanesthetic carbon dioxide tensions in this group.[5]

Patients in the lateral group* were induced with 80 volumes per cent nitrous oxide and 20 volumes per cent oxygen followed by endotracheal ether-oxygen maintenance in a closed system with "circle" soda lime filter. Patients in the prone (Overholt) position were intubated under topical anesthesia and placed in the prone position. General anesthesia was then induced with cyclopropane and oxygen. Anesthesia was maintained with ether and oxygen. The respiration was assisted actively by the hand-bag-squeeze method in all patients in the lateral position. An important limiting factor in the extent to which assistance can be given to the respiration is how much mechanical disturbance the surgeon can tolerate during his work within the chest. In the prone and supine patients ventilation was not assisted.

The Biochemical Determinations and Calculations were carried out as described in our earlier papers.[1-3]

RESULTS AND DISCUSSION

The data are presented in Tables I to V, and are summarized in Table VI. It is evident that elimination of carbon dioxide in the prone position *without* assistance of respiration was as good in the case of lobectomies and better in the case of pneumonectomies than in the lateral position with bag squeezing or machine (see Table VIII) assistance to respiration. Not enough patients have yet been studied in the supine position for adequate evaluation; but it appears that patients on their backs as well as on their bellies (Overholt) are in a more favorable position for the natural excretion (unassisted ventilation) than they are in the lateral position.

Thus *position is a prime factor in the* rise in carbon dioxide during *thoracic* surgery. We are unable to agree with Taylor and Roos[9] that this rise is due to the action of ether on the respiratory center. In the first place, ether is a respiratory stimulant, not a depressant as far as minute volume of respiration goes, at the levels of anesthesia needed for thoracic surgery. Second, no such carbon dioxide accumulation need occur in non-thoracic patients under ether. We[2] showed in twenty patients undergoing surgery in other parts of their bodies but during ether anesthesia and in the supine position that there was no accumulation of carbon dioxide. This has more recently been confirmed in poor risk patients with advanced cardiac, respiratory, and metabolic disease.[4] Actually, these patients[2] blew off carbon dioxide and the pCO_2 was a little below normal at the end of the operation, illustrating the stimulant effect of ether on the respiration.

Nor can we agree with Gibbon and his associates[6] that the rise in carbon dioxide tension is "largely a result of the open thorax, and not the lateral position upon the operating table." In the cases presented here the pleura was open in the prone and in the supine cases, yet without any such tendency for the pCO_2

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*See our previous paper[3] for a full description of placement of these patients.

TABLE I. LOBECTOMY, LATERAL POSITION—ASSISTED RESPIRATION

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
M	48	Rt. U. Lobect.	230	160	7.43*	39	26.4	25.0		(28)	+0.8	25.8	+1.8
					7.37*	34	20.6	19.8		(28)	−0.8	19.0	
M	51	Lt. L. Lobect.	145	95	7.43	41	28.3	27.0	14.3	28.3	+0.8	27.8	+3.7
					7.35	46	27.0	25.2	10.0	22.6	−1.1	24.1	
F	43	Rt. M. Lobect	245	180	7.40	43	25.5	24.0	12.2	25.6	0	24.0	+1.3
					7.40	37	23.9	22.7	9.4	21.7	0	22.7	
F	51	Rt. L. Lobect.	183	95	7.40	44	28.3	26.7	10.8	23.8	0	26.7	+1.2
					7.38	45	27.6	26.0	10.9	23.8	−0.5	25.5	
M	25	Rt. L. Lobect.	180	120	7.42	44	29.2	27.5	12.4	25.9	+0.5	28.0	+1.5
					7.38	47	28.5	27.0	12.2	25.6	−0.5	26.5	
M	60	Rt. M. Lobect.	134	75	7.37	47	28.1	26.4	14.3	28.3	−0.8	25.6	+3.1
					7.32	49	26.2	24.8	14.6	29.1	−2.3	22.5	
F	20	Lt. L. Lobect.	300	225	7.35	49	28.5	26.8	13.4	27.3	−1.4	25.4	+0.8
					7.30	56	29.0	27.1	11.8	25.1	−2.5	24.6	
M	67	Rt. U. Lobect.	115	60	7.45	39	28.0	26.6	13.1	26.9	+1.4	28.0	+1.4
					7.43	40	27.0	25.8	12.9	26.5	+0.8	26.6	
M	62	Rt. U. Lobect.	255	170	7.41	50	32.6	30.9	14.0	28.2	+0.3	31.2	+3.0
					7.20	87	36.4	33.8	13.8	27.8	−5.6	28.2	
F	45	Rt. M. and L. Lobect.	217	155	7.45	40	28.9	27.5	14.1	28.4	+1.4	28.9	+7.9
					7.40	35	22.0	21.0	14.1	28.4	0	21.0	
Average	49		200	134	7.41	44	28.4	26.8	13.9	27.0	+0.3	27.1	+3.0
					7.35	48	26.8	25.3	12.2	27.0	−1.3	24.1	

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE II. LOBECTOMY, PRONE (OVERHOLT) POSITION—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	53	Rt. M. Lobect.	95	50	7.30*	48	24.7	23.0	14.4	28.7	−2.9	20.1	
					7.30*	52	26.8	25.0	14.9	29.3	−2.9	22.1	−2.0
M	61	Rt. U. Lobect.	150	70	7.35	50	28.6	27.0	15.1	29.7	−1.5	25.5	
					7.30	49	25.5	24.0	14.4	28.7	−2.9	21.1	+4.4
F	35	Lt. U. Lobect.	155	85	7.28	54	27.0	25.2	11.7	24.9	−3.0	22.2	
					7.23	56	24.6	22.9	12.1	25.4	−4.3	18.6	+3.6
F	36	Rt. L. Lobect.	130	70	7.45	28	20.0	19.3	12.2	25.6	+1.3	20.6	
					7.40	37	23.5	22.2	11.6	24.8	0	22.2	−1.6
M	55	Rt. U. Lobect.	190	130	7.37	48	29.1	27.3	14.3	28.3	−0.8	26.5	
					7.33	46	25.2	23.6	13.7	27.6	−1.9	21.7	+4.8
F	54	Rt. M. Lobect.	130	80	7.33	41	22.3	21.1	13.8	27.9	−2.0	19.1	
					7.30	45	23.4	22.0	11.5	24.3	−2.4	19.6	−0.5
M	73	Rt. U. Lobect.	145	100	7.41	43	28.5	27.0	14.4	28.7	+0.3	27.3	
					7.33	47	25.7	24.0	10.0	22.6	−1.6	22.4	+4.9
F	50	Rt. M. Lobect.	80	40	7.38	45	27.8	26.0	12.2	25.6	−0.5	25.5	
					7.18	72	28.8	26.2	6.8	18.0	−4.0	22.2	+3.3
M	43	Rt. L. Seg. Lobect.	154	80	7.30	48	24.5	23.0	14.9	29.3	−2.9	20.1	
					7.27	51	24.5	23.0	14.6	29.0	−3.8	19.2	+0.9
M	50	Seg. Lobect.	130	80	7.30	52	26.9	25.0	14.9	29.3	−2.9	22.1	
					7.33	45	24.6	23.1	14.8	29.3	−2.1	21.0	+1.1
M	59	Rt. M. Lobect.	145	65	7.33	48	26.3	24.8	14.2	28.3	−1.3	23.5	
					7.27	47	22.5	21.0	14.1	28.4	−3.7	17.3	+6.2
M	58	Rt. U. Lobect.	205	160	7.35	52	29.8	27.9	13.0	26.8	−1.3	26.6	
					7.25	66	30.7	28.4	12.9	26.7	−4.0	24.4	+2.2
Average	52		142	84	7.35	46	26.3	24.7	13.8	27.8	−1.5	23.2	
					7.29	51	25.5	23.8	12.6	26.2	−2.8	21.0	+2.2

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE III. PNEUMONECTOMY, LATERAL POSITION—ASSISTED RESPIRATION

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
M	70	Lt. Pneumonect.	245	165	7.46*	39	28.3	26.7	12.2	25.6	+1.3	28.0	+4.8
					7.25*	65	30.2	28.0	12.2	25.6	−3.8	24.2	
M	63	Rt. Pneumonect.	220	155	7.40	37	24.0	22.8	11.8	25.1	--	22.8	+0.6
					7.40	37	23.3	22.2	12.2	25.6	--	22.2	
M	56	Rt. Pneumonect.	240	130	7.40	40	25.6	24.1	12.8	26.4	--	24.1	+1.1
					7.25	63	20.1	27.0	13.1	26.9	−4.0	23.0	
M	63	Rt. Pneumonect.	100	115	7.27	56	27.2	25.2	14.6	29.1	−3.8	21.4	−1.4
					7.27	59	28.3	26.4		(28)	−3.6	22.8	
M	16	Lt. Pneumonect.	118	60	7.37	46	27.7	26.0	15.2	30.0	−0.9	25.1	+6.9
					7.30	45	22.8	21.2	15.0	29.5	−3.0	18.2	
M	56	Lt. Pneumonect.	275	205	7.37	47	28.3	26.8	12.2	25.6	−0.8	26.0	+8.2
					7.22	57	24.5	22.8	13.6	27.5	−5.0	17.8	
M	57	Rt. Pneumonect.	240	165	7.42	36	24.1	23.0	12.8	26.4	+0.5	23.5	+7.6
					7.18	60	23.6	21.8	13.0	26.7	−5.9	15.9	
M	54	Lt. Pneumonect.	180	130									
					7.20	74	31.0	28.8	14.3	28.6	−5.7	23.1	
Average	54		202	142	7.38	43	26.5	24.9	13.1	26.9	−0.5	24.4	
					7.25	58	26.6	24.8	13.3	27.2	−3.9	20.9	+3.9

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE IV. PNEUMONECTOMY, PRONE (OVERHOLT) POSITION—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	62	Rt. Pneumonect.	150	100	7.33*	46	25.2	23.6		(28)	–2.0	21.6	
					7.27*	61	29.5	27.5		(28)	–3.6	23.9	–2.3
F	34	Lt. Pneumonect.	120	70	7.35	41	23.6	22.1	12.8	26.4	–1.3	20.8	
					7.30	45	22.5	21.1	12.3	25.8	–2.6	18.5	+2.3
F	29	Lt. Pneumonect.	115	70	7.37	40	23.8	22.3		(28)	–0.8	21.5	
					7.30	39	20.3	19.0		(28)	–2.8	16.2	+5.3
F	29	Lt. Pneumonect.	175	115	7.40	37	23.5	22.2	11.4	24.5	0	22.2	
					7.35	48	27.4	25.8	9.9	22.4	–1.1	24.7	–2.5
M	57	Lt. Pneumonect.	115	65									
					7.32	52	27.7	25.9	14.2	28.5	–2.3	23.6	
F	40	Lt. Pneumonect.	105	65	7.33	40	22.1	20.9	13.7	27.8	–1.9	19.0	
					7.28	46	22.6	21.1	13.6	27.5	–3.3	17.8	
M	34	Lt. Pneumonect.	145	75	7.36	41	24.1	22.8	11.7	24.9	–1.0	21.8	+1.2
					7.36	40	23.1	21.9	15.2	30.0	–1.2	20.7	
F	32	Lt. Pneumonect.	145	70	7.38	42	25.6	24.1	13.3	27.3	–0.5	23.6	+1.1
					7.33	44	24.3	23.0	12.2	25.6	–1.8	20.2	
F	28	Lt. Pneumonect.	60	45	7.35	44	25.2	23.9	11.3	24.3	–1.2	22.7	+3.4
					7.30	61	31.6	29.5	11.7	24.9	–2.5	27.0	
M	62	Rt. Pneumonect.	90	55	7.41	36	23.6	22.3	13.3	27.2	+0.3	22.6	–4.3
					7.30	47	24.4	22.9	13.8	27.9	–2.8	20.1	
M	52	Lt. Pneumonect.	160	120	7.40	37	23.9	22.5	12.5	26.1		22.5	+2.5
					7.38	38	23.0	21.7	12.8	26.5	–0.5	21.2	
F	38	Lt. Pneumonect.	105	45	7.34	48	27.0	25.4	12.0	25.4	–1.5	23.9	+1.3
					7.29	48	24.6	23.0	11.9	25.3	–2.8	20.2	+3.7
Average	41		124	75	7.37	41	24.3	22.8	12.4	26.0	–0.9	22.0	
					7.32	47	25.1	23.5	12.8	26.4	–2.3	21.2	+1.1

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE V. MISCELLANEOUS PROCEDURES, OPEN CHEST, SUPINE—NOT ASSISTED

SEX	AGE (YR.)	OPERATION	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	PH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	$[HCO_3^-]$ (MM./L.)	HEMOGLOBIN (GM. %)	β	CORRECTION	$[HCO_3^-]$ CORRECTED TO PH 7.40	CHANGE IN FIXED ACID
F	60	Lt. Pneumonect.	225	145	7.35* 7.35*	48 42	27.3 24.2	24.8 22.8	14.9 14.8	29.3 29.1	−1.5 −1.5	23.3 21.3	+2.0
F	65	Exploration (Inoperable)	(130)	(60)	7.38 7.37	42 43	25.4 25.8	24.0 24.4	12.9 12.9	26.7 26.7	−0.5 −0.8	23.5 23.4	+0.1
F	38	Rt. M. Lobect.	120	65	7.41 7.27	40 (57)	26.6 (27.8)	25.1 (25.8)	14.5 13.9	28.9 28.1	+0.3 −3.7	26.9 (24.1)	(+2.8)
M	54	Rt. M. Lobect.	140	70	7.39 7.31	43 53	27.3 28.3	25.6 26.4	16.1 15.0	31.2 29.6	−0.3 −2.7	25.3 23.7	+1.6
F	52	Lingulectomy	110	60	7.40 7.35	46 45	29.8 25.7	28.0 24.2	15.2 14.0	29.9 28.6	0 −1.4	28.0 22.8	+5.2
F	32	Exc. Pericard. Cyst	52	22	7.29	45	22.7	21.3	12.8	26.7	−2.9	18.4	
M	63	Lt. Pneumonect.	170	120	7.41 7.45	44 36	29.0 25.5	27.3 24.3	12.6 12.0	26.2 25.4	+0.3 +1.3	27.6 25.6	+2.0
Average	52		135	77	7.39 7.34	44 46	27.6 25.7	25.2 24.2	14.4 13.6	28.7 27.7	−0.3 −1.7	25.8 22.8	+2.3

*Upper line, preanesthetic; lower line, near end of anesthesia.

TABLE VI. EFFECT OF POSITION ON CARBON DIOXIDE TENSION

	PATIENTS (NO.)	AGE (YR.)	PLEURA OPEN (MIN.)	pH	SERUM CO_2 (MM./L.)	CO_2 TENSION (MM. HG)
Lobectomy Lateral position* Not assisted	8	34	96	7.51 7.22	25.9 26.2	33 67
Lobectomy Lateral position Assisted	10	49	134	7.41 7.35	28.4 26.8	44 48
Lobectomy Prone (Overholt) Not assisted	12	52	92	7.35 7.29	26.6 25.5	46 51
Pneumonectomy Lateral position* Not assisted	5	38	122	7.48 7.09	26.6 26.9	35 98
Pneumonectomy Lateral position Assisted	8	54	142	7.38 7.26	26.5 26.6	43 58
Pneumonectomy Prone (Overholt) Not assisted	12	41	75	7.37 7.32	24.3 25.1	41 47
Miscellaneous, Open Thorax Supine Not assisted	7	52	77	7.39 7.34	27.6 25.7	44 46

*Beecher, H. K., and Murphy, A. J.[1]

TABLE VII. PNEUMONECTOMIES, PRONE, NOT ASSISTED (CHANGES OBSERVED DURING INDUCTION, OPENING OF PLEURA, ETC.)

PATIENT	ANESTHESIA TIME (MIN.)	PLEURA OPEN (MIN.)	pH	CO_2 TENSION (MM. HG)	SERUM CO_2 (MM./L.)	
H. B.	0	---	7.40	37	23.9	Preanesthetic
	20	---	7.30	41	21.3	Prone, no surgery
	(60)	(20)	7.32	45	24.5	Chest open
	160	120	7.38	38	23.0	Left lung out, chest open
L. D.	0	---	7.34	48	27.0	Preanesthetic
	30	---	7.28	53	26.4	Prone, no surgery
	75	15	7.27	49	23.9	Chest open
	105	45	7.29	48	24.6	Left lung out, chest open
	160	100	7.28	49	24.2	Chest closed

to rise. The only difference is the interference with the respiration produced by position.*

With the effect of position on the accumulation of carbon dioxide clearly established, we can then turn to a consideration of the best way to prevent the accumulation of it. Gibbon and his associates reported on thirteen patients undergoing pneumonectomy.[6] Notwithstanding artificial ventilation by machine four of the thirteen more than doubled their carbon dioxide tension; they showed a very high carbon dioxide tension, that is, above 90 mm. Hg at the end of operation. The levels in four patients out of thirteen are very high indeed and presumably dangerous. Their average carbon dioxide tension was 74 mm. Hg. Using hand assistance to respiration (bag squeezing) we had only one patient this high and no other above sixty-five. Our average value for carbon dioxide tension in the assisted pneumonectomy group was 58 mm. Hg (see Table VIII). None of Overholt's pneumonectomies carried out in the prone position showed as much accumulation of carbon dioxide, *without any assistance to respiration.* We are obliged to conclude, on the evidence to date, that assistance to the respiration by the simple bag squeezing method such as we used is at least as good and apparently somewhat better and more dependable than that by machine, and that the prone and possibly supine positions are better than either.

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*Serial determinations obtained during two of the prone pneumonectomies are presented in Table VII. These were particularly poor risk patients, and at no time while the pleura was open did either have a carbon dioxide tension above 50 mm. Hg, the accepted upper limit of normal. There was no change in carbon dioxide tension in samples drawn just prior to opening the pleura and just after opening the pleura.

TABLE VIII. COMPARISON OF MANUAL WITH MACHINE ASSISTANCE OF RESPIRATION (PNEUMONECTOMIES, LATERAL POSITION)

	CASES	PLEURA OPEN (MIN.)	pH	SERUM CO_2 (MM./L.)	CO_2 TENSION (MM. HG)
Gibbon et al. (Machine)	13		7.22	26.8	74
Taylor and Roos (Manual)	6	105	7.21	25.7	67
Beecher et al. (Manual)	8	142	7.26	26.6	58

SUMMARY

Work by others has confirmed our original finding that carbon dioxide accumulates to dangerous levels during surgery in the open thorax. This accumulation certainly is related to position of the patient when these patients have an open pleura, it is due to impairment of respiration, of ventilation, of the lungs. It can be, if not entirely eliminated, at least so minimized as to make it of no consequence by nothing more than choice of a favorable position. On the other hand, the simple expedient of bag squeezing by hand to assist the respiration gives dependable and relatively safe protection, at least as good as and apparently better than artificial ventilation by the more complex machine.

The prone and the supine positions are most favorable for the natural excretion of carbon dioxide, but let us say once again it is not our view that surgeons operating in the thorax need adopt either the prone or the supine position. They can choose whatever position that best suits them and suits best the surgery to be done, and rest confident that simple bag squeezing by hand will protect their patient, if they choose the lateral position.

In conclusion, we have shown the existence in thoracic surgery of a real and a common danger. This has been confirmed by others, and we have shown that it can be overcome or at least effectively minimized by simple means. The use of these simple measures is extremely important.

We wish to acknowledge our indebtedness to Dr. Richard H. Overholt of the Deaconess Hospital, Boston, and to Dr. Edward D. Churchill and Dr. Richard H. Sweet, of the Massachusetts General Hospital, Boston, for their permission to study blood gases in their cases.

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DISCUSSION

DR. H. C. MAIER, New York.—Dr. Beecher stated, I believe, that in their series acidosis occurred only during thoracic operations, especially in the lateral position. We, however, have even encountered acidosis in some abdominal cases operated upon in the supine position with ether anesthesia. One can naturally say that this is because the anesthetist was not adequately ventilating the lungs, and such criticism is justified. The point I wish to stress, however, is that it is my impression that as ether anesthesia is employed throughout the country there are many instances in many clinics, the same as in ours, where acidosis occurs due to depressed respirations. Of course the problem of respiratory acidosis is a much more important one during thoracic procedures with an open chest.

(Slide.) This first slide indicates the pH and CO_2 tension changes in a patient undergoing lobectomy and decortication in the lateral position. I would like to emphasize at this point that we feel strongly that every anesthetist, every anesthetic technique, as well as the anesthetic agents, may cause certain variations. In this patient, as is not infrequently true in our cases, we tended to get the patient fairly deep before intubation was done in order to have it performed in a smooth manner from a technical standpoint. As a result in this case we had respiratory acidosis early, although the oxygen saturation stayed quite normal throughout operation. So here we have an instance in which before the patient was turned in the lateral position we had a lower level of the pH than at any time throughout the intrapleural part of the operation. This shows the variations that may occur.

(Slide.) This chart records the pH and CO_2 changes during an operation for mediastinal lymphosarcoma performed through a lateral approach in a somewhat emphysematous individual. Here again we had a marked drop in pH before we turned the patient into the lateral position, due to decreased ventilation, and in this patient too there was no hypoxia at any time. Then the anesthetist gave more adequate ventilation while in the lateral position and corrected the respiratory acidosis, but then another drop in pH occurred later due to the combination of open chest, lateral position, emphysema, and less adequate ventilation. Rapid improvement in the acidosis occurred at the end of the operation in this patient who had no secondary circulatory disturbance.

(Slide.) Here is a case of abdominal gastrectomy with ether anesthesia showing a drop in pH. One can correctly say that this acidosis is due to depressed respirations and inadequate ventilation. But the point is that it did occur in this patient, although there was apparently clinically satisfactory anesthesia with normal oxygen saturation throughout.

(Slide.) This slide shows the pH plotted against length of time from the beginning to the end of a miscellaneous group of operations. You will note that in the absence of any circulatory disturbance occurring during operation there is a rather rapid correction of respiratory acidosis at the end of the operation. Determinations made only at the beginning and the end of operation may give us very little information about what may occur in between these times. If we study this problem we have to get repeated readings throughout the procedure.

DR. EVARTS A. GRAHAM, St. Louis.—I had no intention whatever of discussing this paper when I came here, but I would like to call attention to one or two very simple things. We are constructed in such a way that the act of inspiration consists, at least partly, of elevation of our ribs and expansion of the thorax forward by that means. If we lie on the chest in the prone position without any assistance in getting air into the lungs, with each inspiration we have to raise the trunk off from whatever it is we are lying on. In other words, our respiratory muscles have to do much harder work than they had to do before. If we lie on one side we can breathe more comfortably than if we lie flat on the chest, and measurements of vital capacity in a normal individual will show that fact. However, if, with the patient in the lateral position, one opens one side of the chest, and puts that part of the chest out of commission more or less as a respiratory organ, then the unopened side of the thorax has to lift the trunk with each inspiration, and is handicapped by not having the help of its fellow on the other side, and so of course without artificial assistance again respirations are going to be seriously embarrassed. If we lie on the back, all we have to do with inspiration is to enlarge the front part of the thorax; we do not have to lift the trunk with each inspiration. Therefore, from the standpoint of an ideal position to minimize handicaps to inspiration, it naturally follows that the best is on the back—the supine position. The conditions are still further improved, as judged by the vital capacity, if the trunk and head are elevated to an angle of about 45 degrees. In any condition, however, in which respiration is embarrassed by fluid, by weak muscles, low vital capacity, anesthesia—whatever you wish—it is natural that we ought to protect the body as much as possible by giving artificial assistance.

It seems to me that much of this discussion about whether or not a patient should be operated on in the lateral position, supine position, or prone position, comes down chiefly to the question of—are you going to see to it that the patient ventilates his lungs properly? That is really the problem.

During our work on the Empyema Commission in World War I we did many experiments on ourselves, that is, on normal people and on sick people as well, and we thought that we demonstrated pretty satisfactorily that the prone position, of all of them, is the worst from the standpoint of ventilating the lungs automatically, spontaneously, if the patient is lying on a hard surface, because, as I have just said, with each inspiration he has to lift his whole trunk every time he breathes. Of course Dr. Overholt's table makes the problem a different proposition; it is not the same thing at all. But it all gets back to the fundamental proposition of ventilation, and I think position is much less important than to bear in mind the fact that it is ventilation that is needed.

DR. PAUL W. SANGER, Charlotte, N. C.—I think Dr. Beecher has shown convincing evidence that there is an increased CO_2 tension with the patient in the lateral position, and the reason we do not get into trouble more often must be because the safety factor is pretty large. There are a few conditions, however, in which we cannot depend on the safety factor. We think there are four such conditions, namely: (1) when there is a case with anticipated large blood loss when it is recognized beforehand that the lung is adherent to the chest wall; (2) when the patient is acutely ill; (3) when the vital capacity is low, especially in emphysematous patients; and (4) when we cannot depend on the anesthesia.

In these four groups of patients we are obliged to operate with the patient in the supine position. We have found it expedient to do an anterior thoracoplasty with the removal of the first five ribs to the posterior axillary line, also including their respective costal cartilages. By using the anterior approach, after having removed the ribs, we find that the mediastinal structures can be isolated, ligated, and severed very easily before extreme hemorrhage is created by avulsing the adherent lung from the chest wall. Thereupon the bleeding can be readily controlled by pressure. The postoperative emphysema problem, which has been discussed this morning, we feel is adequately controlled by the anterior removal of the first five ribs. The mere position of the patient reduces the contralateral infection factor, and we think the partial thoracoplasty minimizes the postoperative formation of bronchopleural fistula and empyema. In the seventeen patients on whom we have used this procedure, we have had no symptoms suggestive of an increased CO_2 tension syndrome or other uncontrollable complications.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—I think we will all agree that the work of Dr. Beecher has contributed to our total knowledge of the problem of how best to maintain ventilation during intrathoracic procedures. Dr. Graham mentioned a very important point which was, in effect, that although laboratory studies do seem to indicate that one position might favor another, when we are confronted with the problem of getting the sick person well each surgical team should select that type of approach which will provide the best operation in its hands. There is an advantage for a particular surgical team to develop a more or less routine type of approach that can be carried out with dispatch and convenience. In that way, you add to the safety factor.

Originally, in working with the prone position, we had in mind improving the drainage from the lung as well as improving ventilation on the contralateral side. Then we found that the most serious-risk patient, the patient with the wettest lung, tolerated the open chest better in that position than the good-risk patient with a dry lung did in the lateral position. We came to the conclusion that if this position was of advantage to a bad-risk case, it ought to add to the safety in the management of the good-risk case. Another advantage of the prone position is that one may want to do something more than just excise a lung and, in the supine position, it is a little difficult to carry out a concomitant thoracoplasty if a simultaneous thoracoplasty is contemplated at the time of resection. Again, in some instances where the lung is densely bound down or is involved in a large tumor mass, it is important to have a wide exposure. Then, an approach from the posterolateral aspect has great advantage. There are many factors which enter into the equation, and no one will quarrel with the general proposition that each surgical team must select that particular position which will provide for its patient the greatest margin of safety.

DR. JOHN H. GIBBON, Jr., Philadelphia.—I think we are all agreed that when the pleura is open ventilation is very important. I think it would be nice to state before this Association that our Honorary Member, Clarence Crafoord, was one of the first, ten years or more ago, to emphasize the importance of pulmonary ventilation under these conditions. Also, Drs. Beck and Mautz were among the first thoracic surgeons in this country to appreciate Crafoord's work.

I think Dr. Beecher is to be congratulated upon being the first to report a carefully studied series of patients demonstrating the danger and the everpresent hazard of accumulation of CO_2, with an increase of CO_2 tension and a drop in pH. I think he has also demonstrated that it is probably more difficult to provide adequate ventilation in the lateral position. However, I am not yet convinced that we cannot provide adequate ventilation in that position.

Another thing which I think should be borne in mind is that with these relatively small series of cases which we all have, there is quite a large individual variation. Both Dr. Beecher and I have reported averages and have also given individual cases. But we have had patients, as Dr. Beecher has I know, that have been operated upon in the lateral position without any respiratory acidosis.

Why is it that is some cases acidosis develops in spite of apparently adequate ventilation? I think very often it is because the patient is emphysematous or has a greatly reduced vital capacity. In other words, we are dealing with the type of patient in whom, probably in any situation, it might be difficult to avoid an increase in CO_2 tension. I think before we come to rigid conclusions about position or method of ventilation—I hold no brief for the mechanical method of ventilation—we should remember that our series are still small, that we must collect more data, and that we must ascertain the facts about these enormous individual variations.

Finally, I might add relative to that point that we did study one patient on whom a pneumonectomy was performed in the Overholt face-down position. This patient had the most severe respiratory acidosis of any in our series despite determined effort to provide adequate ventilation. The citing of this single case again does not constitute evidence that the position was at fault. I think Dr. Beecher has very suggestive evidence that in the prone and supine positions it is easier to avoid respiratory acidosis than it is in the lateral position. I am merely pointing out that there are marked individual variations with the use of any position.

What we all want to be able to do is to recognize the development of respiratory acidosis when the patient is being operated upon. We do intermittent blood sampling now, which necessitates maintaining an indwelling needle in an artery throughout the operation. The sample has to be analyzed, which takes time, and you do not know during the course of the operation whether or not the CO_2 tension is going up and the pH dropping. I think we are all interested in the development of some method of knowing quickly, easily, and continuously the percentage of carbon dioxide in the expired gases during a prolonged operation. I hope someone will develop such a method in the next few years.

DR. LLOYD H. MOUSEL, Seattle.—Dr. Beecher is to be complimented for the fine work he has done on respiratory acidosis.

He spoke about assisted respiration. Perhaps we should mention some of the methods of assisting respiration and compare them with controlled respiration. There is a great deal of difference, and certainly we must at times interfere seriously with the lesser circulation when we assist or control this most important function.

In a preliminary study made by Dr. Howard Pierpont a little more than a year ago, pressure readings were made within the pulmonary arteries of dogs during assisted and controlled respiration. The pressure within the pulmonary artery rose markedly in those animals which were being subjected to controlled respiration. In my opinion, Pierpont's observations are of clinical importance. If we control respiration, or assist it too vigorously, it seems reasonable to assume that a pulmonary hypertension will develop. This increased work load, suddenly thrown upon a right heart in which myocardial changes are already present, may result in congestive heart failure or even sudden death during operation. Young patients with normal hearts undoubtedly compensate for the pulmonary hypertension which results from positive pressure breathing. Patients in the older age group ought to be watched carefully. If assisted respiration is necessary the mechanical efforts need to be coordinated with the patient's own efforts to breathe. The amount of pressure exerted on the breathing bag should be limited to that pressure which will assure a sufficient tidal volume of respiration. At the end of the inspiratory phase the pressure within the breathing system should be allowed to drop to atmospheric pressure.

Controlled respiration should be avoided if possible. In case there is need to control respiration for a short period, sustained pressures are to be avoided. Any amount of constant positive pressure within the breathing system will be transmitted to the alveoli. The pulmonary capillary system will be compressed, preventing adequate filling and resulting in increased pressures within the chambers of the right side of the heart.

DR. OSLER A. ABBOTT, Atlanta.—First, I would like to say that the ventilation factor so thoroughly stressed by Dr. Beecher is probably the most important one in this discussion. I wonder if, by any chance, we are missing something else of major importance. There is a difference in the position, both of the lobe upon which we must operate and the adjacent lung structures, according to the position in which the patient is operated upon. The degree of traction upon an adjacent lobe and displacement may well kink the vessels in the hilus, and in some cases partially obstruct pulmonary venous return and set us a temporary state of pulmonary vascular congestion. This kinking is much minimized by the face-down position. There is also the factor of the traction and torsion upon the bronchial hilar structures and their effect upon pulmonary function and bronchospasm.

We used to be members of the lateral position school. It was customary, during the first week or ten days following surgery, to visualize a well aerated residual lobe following lobectomy, but to find rather inadequate breath sounds on auscultation. Following cough these breath sounds would come through in a mildly tubular character. We have felt that this is a good clinical sign of pulmonary dysfunction on the operated side and may be related to resolution of congestion, or even of ecchymosis in the retracted lobe. This situation, of diminished to absent breath sounds over a residual, roentgenologically demonstrably expanded lobe, does not occur following the face-down position, and we feel it is due to the lack of torsion upon the hilar pedicle and, indeed, the lack of handling this lobe almost completely during surgery. We would therefore like to stress that there is more than just the ventilatory factor; there is also the handling of the lobe and the kinking of the vascular pedicle and of the bronchial pedicle to the lung.

Before terminating my remarks, I would like to emphasize the danger of the position of the sternal rest which may be used in the face-down position, particularly with some of the attachments which are used with the usual operating table. The lower sternum may be a remarkably flexible structure and, in a patient with a thin P-A diameter of the chest, there may be a very marked compression of the heart and actually a physiologic cardiac tamponade produced if the sternal rest is placed in the midsternal or lower sternal region. It is very important that this rest be placed so that it impinges upon the sternal notch. This is a good thing for which to look in the patient whose blood pressure falls and pulse pressure seems to contract when placed in the face-down position.

DR. ROBERT H. WYLIE, New York.—Added evidence of this mechanism has been observed by us during the course of operation for mitral stenosis. With continuous electrocardiography we have observed bundle of His and nodal rhythm, and T-wave changes which have been completely obliterated by asking the anesthetist to increase the ventilation.

DR. CLARENCE DENNIS, Minneapolis.—Some of us in Dr. Wangensteen's department in Minneapolis have been working for several years to try to develop a combined pump and oxygenator that would support the entire circulation without the employment of the lungs or heart of the subject in question. One of the major problems which has perplexed us is the problem of elimination of carbon dioxide in our dogs prior to the time the machine is employed. We have had our dogs on one side much of the time and have opened both sides of the pleura, and it has been possible to examine the down side lung, both during and after the procedure, and in those dogs sacrificed some time afterward.

We were not satisfied with any of the methods of maintaining respiration, using the lungs, that were available to us. We therefore developed a respirator which seems to have solved the problem for us. This is not the time to describe this, but I would like to point out that the things that seemed important were these: In the first place, the expiratory phase must be characterized by a sharp drop all the way to atmospheric pressure, as Dr. Mousel emphasized. In the second place, during the inspiratory phase, the lung must be maintained with a plateau rather than a peak of pressure, and we have found that, so far as our dogs were concerned, a plateau of 16 or 17 cm. of water pressure occupying about one-third of the respiratory cycle is enough to keep the down side lung from undergoing compression atelectasis from the weight of the mediastinal structures that lie on the lung during the time the dog is in that position.

DR. BEECHER (closing).—I am grateful to Dr. Graham, Dr. Maier, Dr. Sanger, Dr. Overholt, Dr. Gibbon, Dr. Mousel, Dr. Abbott, Dr. Wylie, and Dr. Dennis for their helpful discussions.

Dr. Maier emphasized the fact that too deep anesthesia is undoubtedly common over the country; I know that is so. One need not use overly deep anesthesia to carry out any kind of surgery needed. Of course the most difficult problem so far as this is concerned is upper abdominal surgery. We have a good deal of blood gas data during upper abdominal surgery and we know that abnormal elevation of carbon dioxide tension is not necessary even here. The acidosis Dr. Maier spoke of midway in his operations is doubtless due to the fact that most anesthetists (and surgeons) like to have their patients much more deeply anesthetized midway in the operation than they do at the end. We hope we have shown that the surgeon operating in the thorax can use whichever position is best for the surgery and best for his own practices. It is obvious that if the surgeon does use the lateral position, his anesthetist must employ some kind of aid to ventilation. I hope I did not sound dogmatic in arguing for the bag-squeeze method over the machine. I am simply recording that, at the present time, the simple bag-squeeze method—and I must say simple methods appeal to me more than complex ones—the simple educated hand is at least as good, so far as we can tell from factual data at present available, as the more complex machine. As Dr. Gibbon pointed out, four of his pneumonectomy patients did show acidosis. Neither are we successful in maintaining the acid-base equilibrium we would like in the pneumonectomy case. Neither method is perfect. We need to know a good deal more about how to perfect these methods. Rises in four out of thirteen is not good enough. Our record is no better and it is not good enough either. The situation could be handled much better if we had some way, as Dr. Gibbon pointed out so well, of detecting when the pCO_2 rises to troublesome levels. Dr. Wylie spoke of his work with Dr. Himmelstein indicating that electrocardiograms are of assistance, and perhaps that will be one way of indicating when the tension is getting too high.

MUCOID IMPACTION OF THE BRONCHI

ROBERT R. SHAW, M.D.*
DALLAS, TEXAS

WITHIN the past four years we have seen ten patients suffering from what seems to be a distinct clinical entity within the lungs that has not been clearly described in medical literature. These patients all had an underlying condition of asthma or chronic obstructive bronchitis. We have used the descriptive term "mucoid impaction of the bronchi" to designate this entity. It is important that it be recognized clinically since its symptomatology and roentgen appearance may closely simulate other diseases—namely, neoplasm, abscess, and tuberculosis—which have an entirely different prognosis.

Mucoid impaction of the bronchi results from a localized accumulation of inspissated mucus in the bronchi. Typically, this impaction occurs in the second order branch bronchi distal to a bifurcation. The impaction grows in size, layer by layer, in a laminated manner to finally dilate the bronchus in which it lies to many times its normal diameter. As the impaction enlarges it compresses the neighboring branch bronchus so that it also accumulates a mucoid mass which enlarges in the same manner. As the bronchial wall is dilated by the mucoid mass it becomes infected. The cartilaginous elements are almost entirely destroyed, and there is an infiltration of lymphocytes and eosinophilic neutrophils into the wall. The characteristic yellowish-white appearance of the normal bronchus changes to a reddish brown. The mucous lining loses its ciliated columnar epithelium and is replaced by squamous epithelium. Beyond the blocked bronchus suppuration occurs. This suppuration may be in the form of an abscess. This abscess, however, is of the nonputrid variety. At times the suppuration is entirely intrabronchial, causing eventually the formation of cystic bronchiectasis. Typically these cysts will be completely filled with purulent secretion. Seldom will air slip by the mucoid masses to occupy space within these cysts. The alveolar spaces which remain intact contain an amorphous eosinophilic staining material which contains numerous plasma cells, lymphocytes, and occasionally giant cells. Commonly the bronchioles are filled with neutrophils and lipophages. Fat stains may reveal fat in epithelial cells of some of the bronchioles and alveoli, and at times the fat is free due to the breakdown of the macrophages.

The plugs of mucus range from 2.5 to 3.5 cm. in length and from 0.9 to 2.3 cm. in diameter. They are a glary, greenish-gray in appearance, and putty-like in consistency. The cut surface reveals a thick, tenacious ropy material. Section of the plugs reveals evidence of lamination.

From the Surgical Service of the Southwestern Medical College of the University of Texas.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

*Clinical Professor of Surgery, Southwestern Medical College.

The most striking feature of the pathology is the gross finding of the paired impactions of mucoid material in major branch bronchi, and the lipoid granulomatous inflammation of the destroyed wall of the involved bronchus and the pulmonary tissue beyond the plugs.

ROENTGEN FINDINGS

The roentgen appearance of mucoid impaction may be quite varied. The mucoid plugs themselves are large enough to be shadow producing (Figs. 4 *A* and 5 *A*). The shadow is characteristically near the hilum of the lung and appears as a V-shaped shadow. As with a hilar neoplasm the shadow cast by the plugs may be masked by the larger shadow due to the suppuration beyond (Fig. 3 *A*). The shadow cast by the suppuration varies. It may be typical of a pneumonitis (Fig. 2 *B*). This may be the reaction actually present in Loeffler's syndrome. Characteristically the suppuration beyond the plugs casts a sharply circumscribed dense shadow which is indistinguishable in appearance from a pulmonary neoplasm (Figs. 2 *A*, 3 *A*, and 6 *A*). If air is able to slip past the mucoid plugs an annular shadow denoting an abscess with a fluid level may be seen (Fig. 7 *B*). In other instances cysts uniform in size with or without fluid levels may be evident (Fig. 5 *A*). Serial roentgenograms may show clearing of one segment of lung only to have the same type of shadow reappear in other distant segments. At times the sharply circumscribed shadows may completely disappear leaving little roentgen evidence of damage to the lung (Fig. 7 *D*). With clearing of the suppuration the shadows cast by the plugs may persist.

CASE REPORTS

In eight of the ten cases being reported the diagnosis was confirmed by examination of the surgical specimen.

CASE 1.—E. C. F., a 54-year-old man, had had frequent intermittent colds and cough for the past twenty-five years. The cough had always been nonproductive. For the past nine months he noticed dyspnea on exertion, weakness, and anorexia. There had been no fever.

Bronchoscopy: (May 11, 1948.) There was thin watery secretion in the trachea, both bronchi, and their branches. Around the dorsal bronchial orifice on the right the mucosa was thickened and inflamed. Purulent material could be seen coming from this bronchus. No neoplastic tissue could be seen. Bronchography showed obstruction of the dorsal bronchus on the right, and cystic bronchiectasis of the anterior segment of the right upper lobe (Fig. 1*B*).

Operation: (June 24, 1948.) Preoperative Diagnosis: Question of bronchiogenic carcinoma. Bronchiectasis anterior segment, right upper lobe. The right chest was opened through the sixth rib bed. There were adhesions between the dorsal lobe and the posterior chest wall and also adhesions in the interlobar fissures, but otherwise the pleural cavity was not obliterated. A hard mass could be felt in the dorsal lobe which was thought to be carcinoma. The bronchus to this lobe was especially hard, and there was no doubt in the operator's mind that a malignancy was present. The dorsal lobe was removed by dissection technique. The anterior inferior segment of the upper lobe was removed in the same manner. Before the chest was closed the specimen was examined, nad it was found that two large plugs of inspissated mucus had greatly dilated the branch bronchi to the dorsal segment. Beyond these dilated bronchi was saccular bronchiectasis with pneumonitis and fibrosis.

Postoperative Course: In the first postoperative check-up film a rounded shadow approximately 4 cm. in diameter was noted in the lower portion of the right chest. (Fig. 1*C*.)

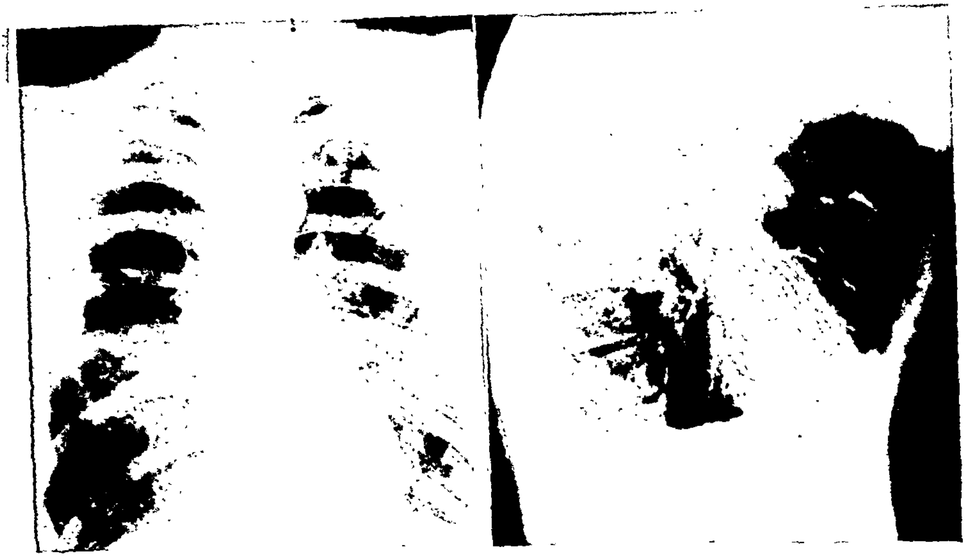
A. B.

Fig. 1, *A* and *B*.—(Case 1) Note diffuse shadow in right hilum. Lateral bronchogram shows saccular bronchiectasis of anterior segment of upper lobe and abrupt block of dorsal bronchus by a discrete shadow-producing lesion. Block was mucoid impaction.

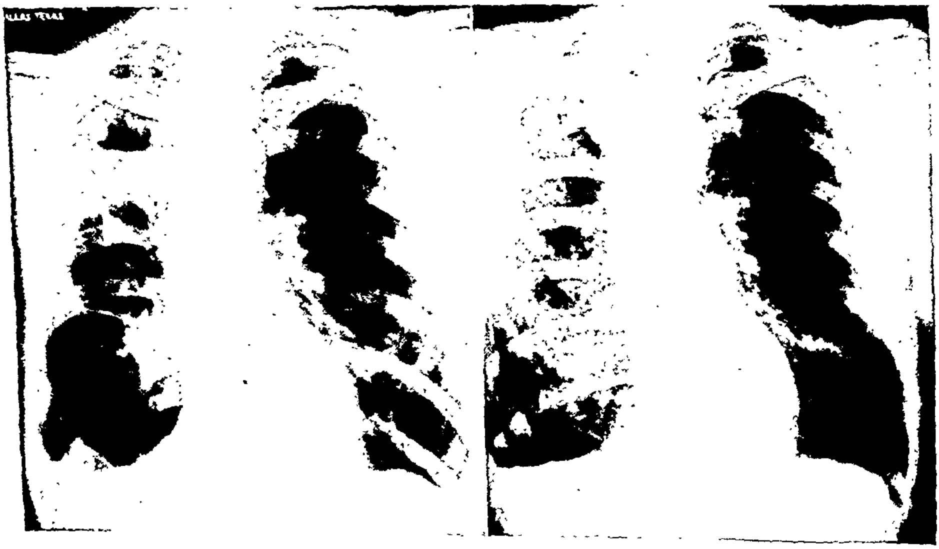
C. D.

Fig. 1, *C* and *D*.—Two months following resection of anterior segment of right upper lobe and dorsal segment of lower. A rounded shadow due to mucoid impaction has appeared which later almost completely disappeared.

The most striking feature of the pathology is the gross finding of the paired impactions of mucoid material in major branch bronchi, and the lipoid granulomatous inflammation of the destroyed wall of the involved bronchus and the pulmonary tissue beyond the plugs.

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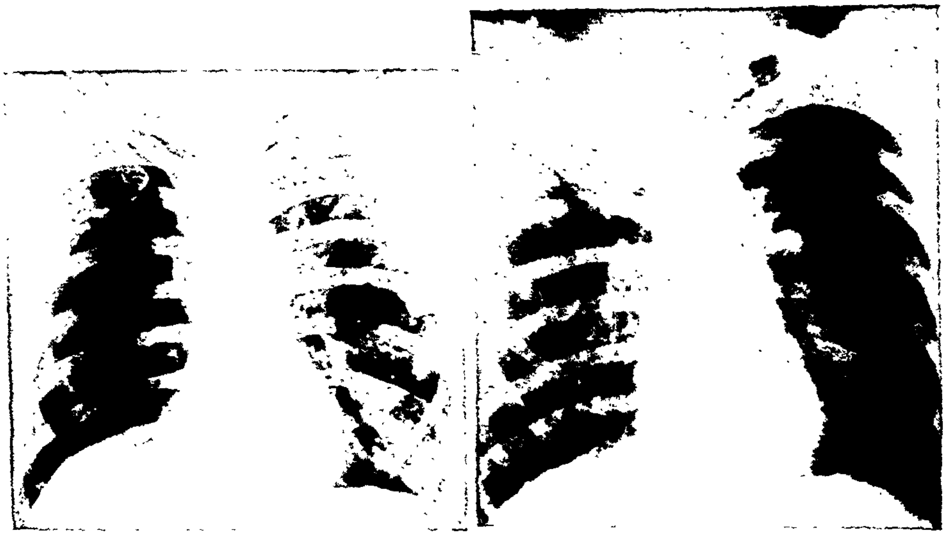
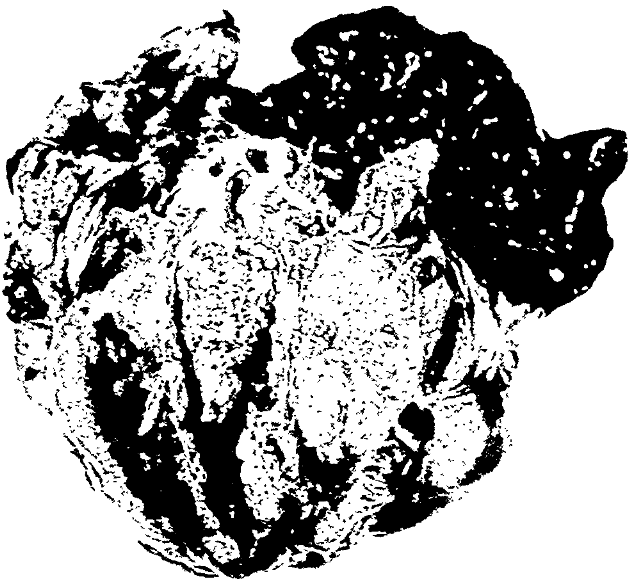
his chest. At that time, roentgenogram showed areas of pneumonitis in both the upper and lower lobes on the left, which indicated that the same process that had been seen in the right upper lobe was starting there (Fig. 2*B*).



A. *B.*

Fig. 2.—(Case 4) *A*, Dense shadow in right upper lobe. Upper lobe lobectomy revealed mucoid impaction with nonputrid abscess, beyond obstructed bronchus. *B*, Six months following lobectomy there was an indication of the same process beginning in left lower and left upper lobes.



C.

Fig. 2, *C*.—(Case 4, J. T.) Right upper lobe. Note large ovoid impaction of mucus dilating and blocking a branch bronchus.

The etiology of this shadow was not determined, and one month later it had disappeared. (Fig. 1*D*.) The patient continued to have cough and expectoration of moderate amounts of thick, tenacious mucus. He did, however, begin to gain weight and strength.

CASE 2.—J. L. B., a 25-year-old physician, had had bronchial asthma all of his life. Skin tests had revealed numerous specific allergies. A roentgenogram of his chest in April, 1947, was perfectly clear. In June, 1947, he had a bout of fever which was diagnosed as atypical pneumonia. At this time in the roentgenogram a shadow was seen in the base of the right upper lobe. He responded well to penicillin, but the shadow in the subapical segment of the right upper lobe never entirely disappeared. This was observed over a period of a year. Since the shadow persisted, and the patient continued to have low-grade fever, multiple hemoptyses, and undue fatigue, right upper lobe lobectomy was advised.

At operation, July 16, 1948, examination of the right upper lobe bronchus revealed gross dilatation of the branch bronchi to the subapical segment of this lobe. Since the bronchus was partially destroyed it was decided that an upper lobe lobectomy would be necessary in order to section the bronchus in normal tissue.

Pathologic Examination: Upon opening the grossly dilated bronchi two large plugs of inelastic soggy, dessicated, gellatinous mucus were found. Beyond the plugged bronchi was cystic bronchiectasis filled with creamy pus.

Postoperative Course: The patient made an uneventful postoperative recovery and stated that he has been more free of the asthma than at any other period in his life.

CASE 3.—E. Z., aged 14 years. One month before admission to Baylor Hospital on Aug. 2, 1947, this boy noticed a slight cough and fever. The cough was nonproductive. The temperature rose to 103° F. He was treated with penicillin, and the temperature soon receded to normal. The roentgenogram of the chest showed a shadow in the right upper lobe which had persisted in spite of the chemotherapy. There was no sputum. This patient had been asthmatic all of his life. On August 12 a right upper lobe lobectomy was done. At surgery, there were found to be dense adhesions between the visceral and parietal pleura over the right upper lobe. There were numerous enlarged lymph nodes in the hilum of the upper lobe.

His convalescence was uneventful, and he has remained well.

Pathology: There was marked induration and firmness throughout 85 per cent of the specimen. On cut surface through the indurated area there was a marked outflow of thick, purulent material from numerous areas. On tracing the bronchi a large impaction of mucoid material was encountered measuring approximately 3½ cm. in length and 1.3 cm. in diameter. This mucoid mass was blocking the bronchus to a major portion of the lobe.

CASE 4.—J. T., aged 45 years, had been troubled with asthma most of his life. He was a painter by trade. Because of fever and malaise a roentgenogram was taken of the chest on April 22, 1949, which showed a dense area in the right upper lobe. (Fig. 2*A*.)

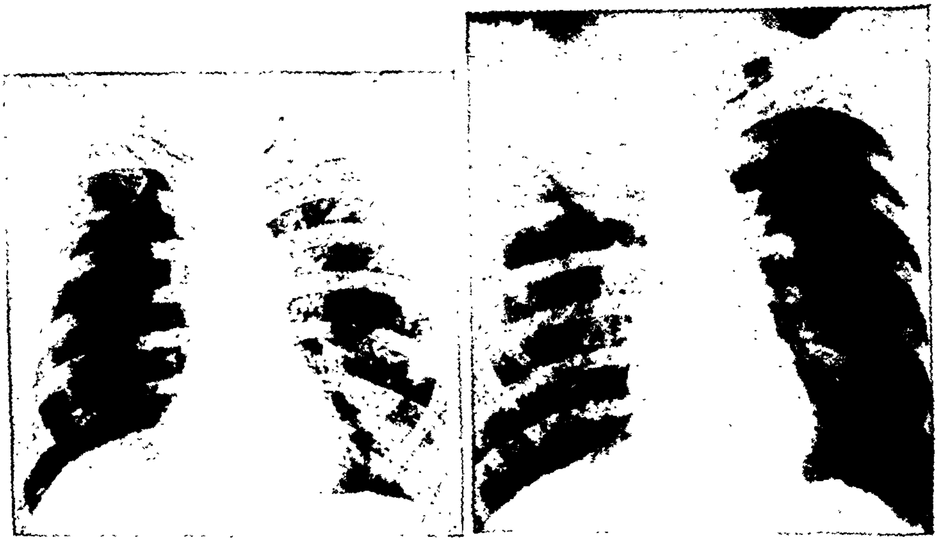
Bronchoscopy: (Nov. 9, 1949.) The mucosa surrounding the upper lobe orifice was hyperemic and edematous. There was no evidence of neoplasm. A sucker tip was passed into the right upper lobe bronchus and a small amount of thick secretion was obtained which was studied for neoplastic cells. This study revealed no atypical cells.

Preoperative Diagnosis: Nonputrid abscess of lung? Bronchiogenic carcinoma.

Operation: (Nov. 10, 1949.) The upper lobe was removed without difficulty by ligation technique. A complete parasympathetic denervation of the remaining portion of the right lung was carried out because of the underlying asthmatic condition.

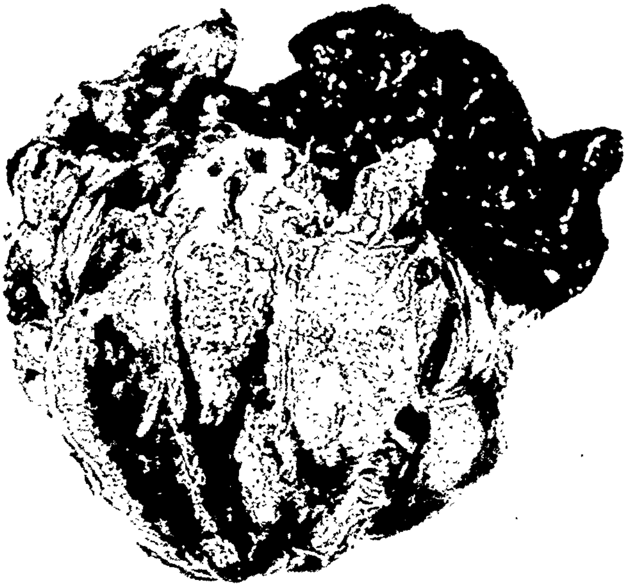
Pathology: The lobe was completely replaced by a spherical mass measuring 10 cm. in diameter. On cross section multiple cavities all filled with greenish purulent material were noted. The major bronchi to the lobe were completely filled and dilated by large, gummy plugs of mucus.

Postoperative Course: The patient made an uneventful recovery from the surgery and returned home. At check-up examination on December 17, it was noted that he was coughing very little. He had gained weight, and the breath sounds seemed clearer on the right than on the left side. In April, 1950, however, he began to have further trouble with his chest. At that time, roentgenogram showed areas of pneumonitis in both the upper and lower lobes on the left, which indicated that the same process that had been seen in the right upper lobe was starting there (Fig. 2*B*).



A. *B.*

Fig. 2.—(Case 4) *A*, Dense shadow in right upper lobe. Upper lobe lobectomy revealed mucoid impaction with nonputrid abscess, beyond obstructed bronchus. *B*, Six months following lobectomy there was an indication of the same process beginning in left lower and left upper lobes.



C.

Fig. 2, *C*.—(Case 4, J. T.) Right upper lobe. Note large ovoid impaction of mucus dilating and blocking a branch bronchus.

Case 5.—Mrs. J. M. was admitted Feb. 15, 1949. This 57-year-old woman had noticed a pain in the left chest which radiated down the left arm for the past year. There had been no cough and no weight loss. She stated that she wheezed when she lay on her left side. She had noticed no fever. The past history was noncontributory except for a questionable coronary attack seven years previously.

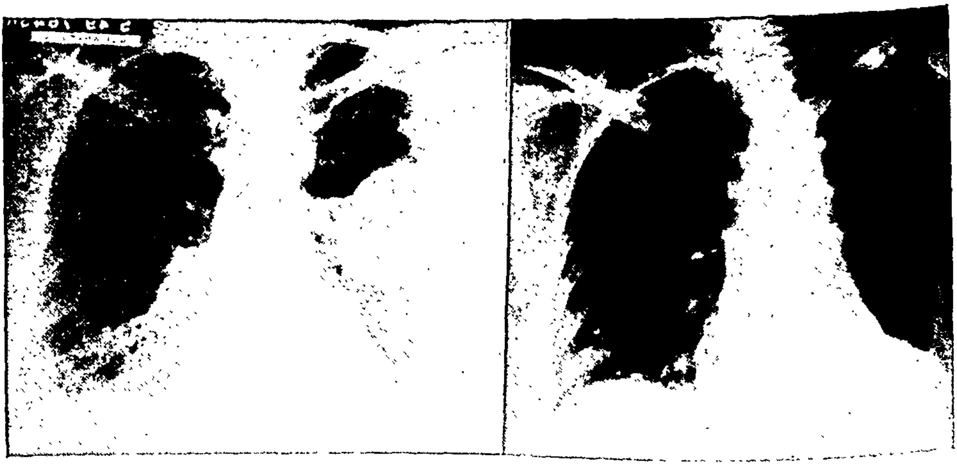
The roentgenogram of the chest showed a large, rounded shadow in the left midlung field (Fig. 3*A*).

A diagnosis of pulmonary neoplasm was made, and surgery was advised.

Operation: (Feb. 16, 1949.) When the left chest was opened the upper lobe was found to be densely adherent to the chest wall. The lower lobe was completely free of adhesions. Since the hilum was uninvolved, only the upper lobe was removed.

The patient's convalescence was uneventful, and she has had no recurrence of similar trouble.

Pathology: Upon sectioning the lobe removed, a large multiloculated space was opened which contained brownish semisolid material. The bronchus supplying this area was greatly dilated by a large mass of inspissated mucoid material. Microscopic examination of this bronchus showed the mucosa to be thin and atrophic and densely infiltrated with inflammatory cells.



A. *B.*

Fig. 3.—(Case 5) Left upper lobe lobectomy done for suspected neoplasm. *A*, Mucoid impaction found. *B*, Eight months following resection chest was clear.

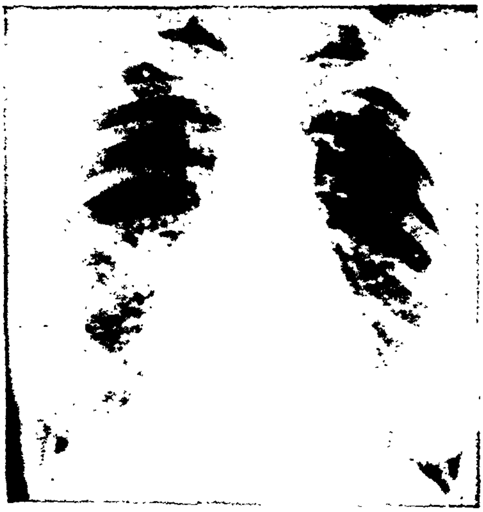
Case 6.—Mrs. J. B. H., aged 53 years, developed virus pneumonia in March of 1949. She had a hard unproductive cough. She stated that she had had previous difficulty with her right lung. She had been asthmatic all of her life. At one time she coughed up a hard plug of material, and this was followed by the coughing up of much pus. Roentgenograms showed destruction of the right middle lobe (Figs. 4*A*, *B*, *C*). Right middle lobectomy was devised.

Preoperative Diagnosis: Mucoid impaction of the right middle lobe bronchi with residual fibrosis.

Operation: The pleural cavity was free of adhesions except over the middle lobe where adhesions were dense and vascular. The middle lobe was removed by dissection technique.

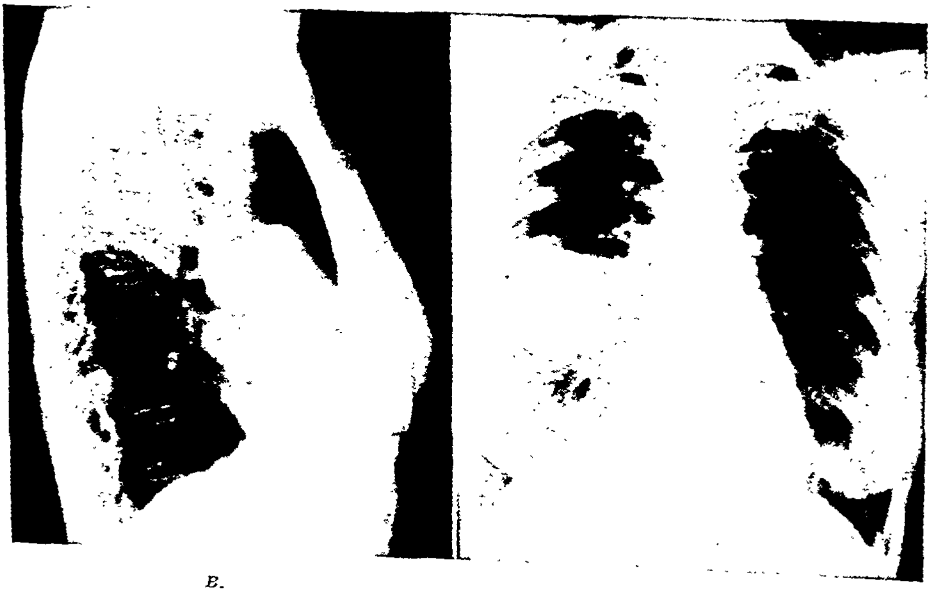
Postoperative Course: Postoperative convalescence was uneventful, and the patient's health has been greatly improved.

Pathology: One branch bronchus of the middle lobe contained a large, oval foreign body of thick, tenacious, mucoid material. It measured 2½ cm. in length by 0.9 cm. in diameter. Sections through other areas revealed similarly dilated bronchi which extended toward the periphery of the lung tissue. These were filled with tenacious mucoid material. Beyond these areas there was bronchiectasis, cylindrical in type. Sections through the gross mucous plug obstructing the bronchus revealed it to be composed of laminated layers of eosinophilic mucoid material.



A

Fig. 4. A.—(Case 6) Note shadows in right hilum cast by mucoid masses dilating the middle lobe bronchi.



B. C.

Fig. 4, B and C.—Same patient five months later. The middle lobe was swollen due to suppuration beyond the obstructed bronchi.

CASE 7.—O. B., aged 19 years, had been sick for over a year with a chronic cough which was productive of small amounts of purulent sputum. Tubercle bacilli could never be found in the sputum. Nevertheless a diagnosis of pulmonary tuberculosis was made, and he spent six months in a tuberculosis sanatorium. It was finally realized that his disease probably was not tuberculosis. Since this patient had been troubled with asthma most of his life, it was suspected that he had mucoid impaction of the bronchi. Exploration of the left chest was advised.

Operation: (March 2, 1950.) The upper lobe was found to be adherent to the anterior chest wall over a rather extensive indurated area. It could be seen that a portion of the indurated area was definitely cystic. The upper lobe was removed.

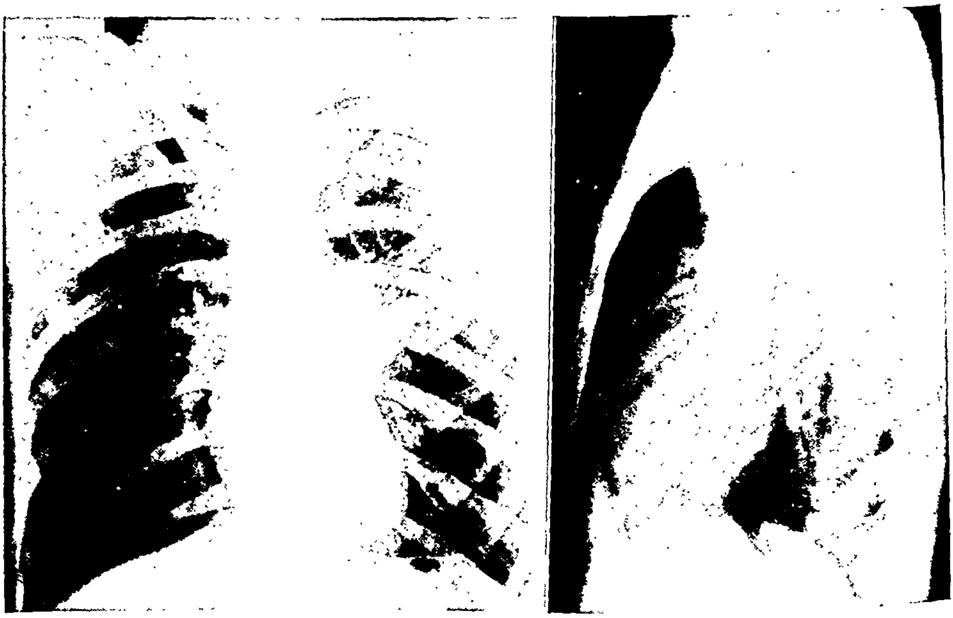


Fig. 5, *A* and *B*.—(Case 7) The lift hilar shadows were due to mucoid impaction. The peripheral shadows proved to be pus-filled bronchiectatic cysts.

Postoperative Course: The postoperative course was marked by excessive bleeding from the parietal pleura which necessitated returning the patient to the operating room for evacuation of a large clot in the pleural space. Following this he made a good postoperative recovery. He was seen again in November, 1950. At this time he stated that he was beginning to have fever again and was not feeling well. A roenegenogram of the chest showed that there was pneumonitis in the right upper lobe, suggesting that the same process was again beginning in a segment of this lobe.

Pathology: Evidence of disease was limited to the posterior and apical segments. The lingula was normal. In the bronchi to these segments there was impaction of thick mucoid material so that the bronchi were dilated to more than three times normal caliber. Beyond these dilated bronchi were cystic areas completely lined with epithelium. These cysts were filled with thick, creamy, mucopurulent material. On microscopic section, there were some areas of the lung near the region of the encapsulated cystic spaces where there were seen a great number of lipophages.

CASE 8.—J. R., a 26-year-old woman, had been an asthmatic since childhood. Her mother had been a chronic asthmatic, and several members of the mother's family had been confirmed asthmatics. At the age of 7 years she had extensive skin testing done to determine a possible allergic background. In 1949, a sharply circumscribed shadow was noticed in the left upper lung field posteriorly (Fig. 6*A* and *B*). This shadow did not change in size or shape over a period of months of observation.

Because of the underlying history of asthma, the general good condition of the patient, and the appearance of the roentgenogram, a diagnosis of mucoid impaction of the bronchi was made in October, 1949. Surgery was not recommended. Due to the fact, however, that there continued to be a suggestion on check-up roentgenograms that a malignancy might be present, it was determined to do an exploration of the left chest.

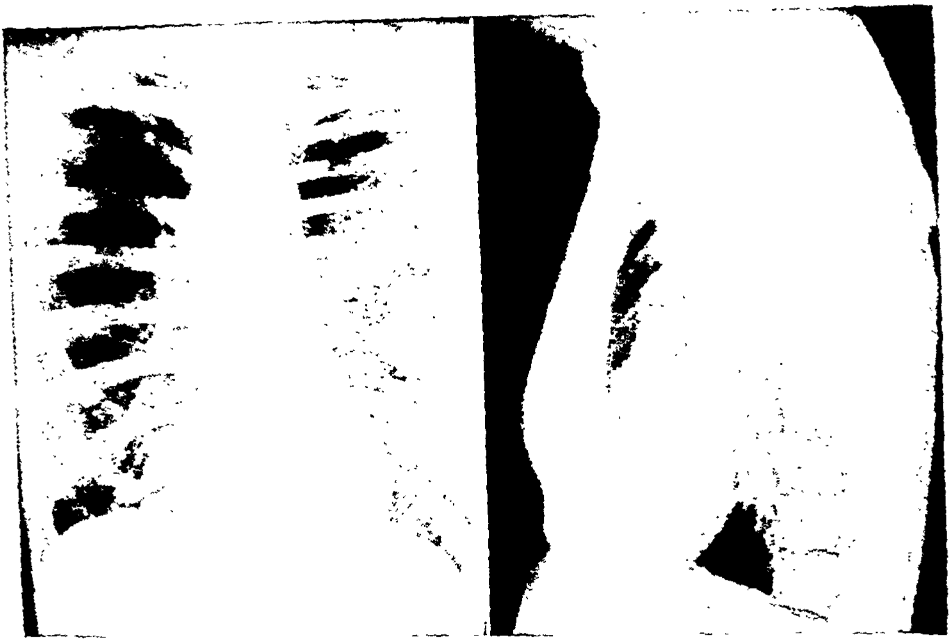


Fig. 6, *A* and *B*.—(Case 8) Sharply circumscribed shadow in left upper lobe due to bronchiectatic cysts beyond bronchi blocked by mucoid impactions.

Operation: On June 26, 1950, the left chest was explored. There were fine avascular adhesions between the cystic area in the left upper lobe and the neighboring left lower lobe and chest wall. There were more vascular adhesions holding the apex of the left upper lobe to the dome of the chest. The cystic areas could be seen on the surface of the subapical division of the left upper lobe, and a hard area could be palpated in which there was mucoid impaction which ran down into the hilum of the lung. The apical and subapical segments of the left upper lobe were removed.

Pathologic Report: Specimen consisted of the upper two-thirds of the left upper lobe. In the bifurcation of the segmental bronchi of this segment of lung were two mucoid plugs. One plug measured 3.1 by 1.5 cm.; the second plug 3.6 by 2.3 by 1.2 cm. These plugs caused the bronchi at the point of bifurcation to become greatly dilated and to have very thin yellowish linings. Distal to the mucoid plugs was a multilocular cavity which measured approximately 4.4 by 3.5 by 2.3 cm. The mucoid plugs had a greenish-gray-white appearance and were puttylike in consistency. The cut surface revealed a thick, tenacious, ropy mucoid material.

The patient's convalescence was uneventful, but she is still subject to attacks of asthma.

Two patients have not had surgery. The history, roentgenogram findings, and course of their pulmonary disease seem to clearly indicate that they have had mucoid impaction of the bronchi.

CASE 9.—J. H., aged 18 years, was hospitalized in September, 1943, because of pneumonia in the right upper lobe. This cleared after a long convalescence, but left shadows in the right upper lung field. In September, 1946, she had an hemoptysis, and has had repeated hemoptyses since that time. There has been no recurrence of the cough and expectoration or fever that marked the initial illness. This patient had been asthmatic almost all of her life. Right upper lobe lobectomy was advised because of the repeated hemoptyses, but this was not accepted by the patient. The patient did return to the office, however, at intervals for check-up examinations. Small hemoptyses persisted, but these never became alarming. In view of the underlying history of asthma and the appearance of the roentgenograms, it was felt that a diagnosis of mucoid impaction of the bronchi was justified. Undoubtedly, there is a cystic bronchiectasis in the apical divisions of the right upper lobe. It may be necessary at a later date to do a resection because of the residual destruction of the lung.

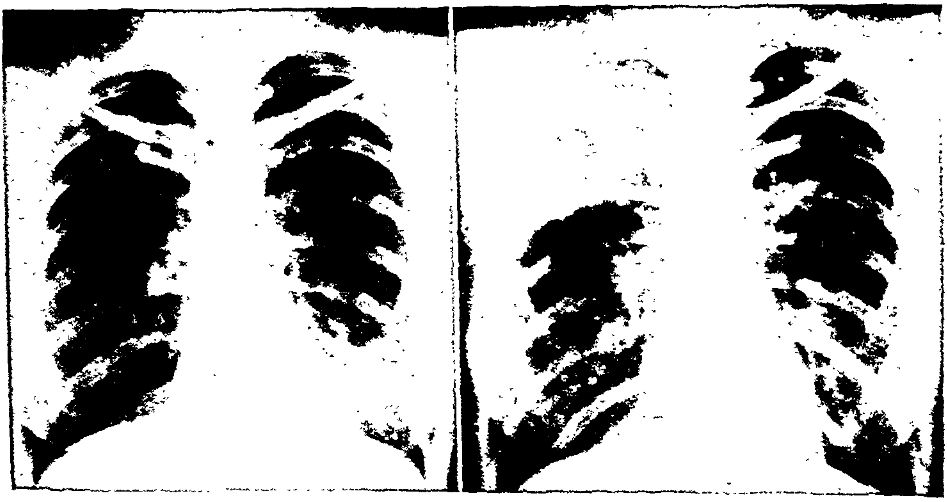
CASE 10.—Mrs. C. W., aged 29 years, had a history of allergy for ten years with asthma and hay fever. As a child she had been considered an asthmatic. In July, 1945, she had what was diagnosed as virus pneumonia. She coughed for several weeks. Penicillin therapy was used at that time both intramuscularly and by inhalation. In February, 1947, an asthmatic attack became severe and prolonged, and was not relieved by adrenalin. A bronchoscopy was done at that time. In November, 1947, she began to cough and has had a rather persistent cough ever since that time. She changed her climate from that of the Panhandle region of Texas to Tucson, Arizona. This did not improve her symptoms greatly, and during that time an abscess developed in her lung. A review of the films brought by the patient showed that she had had, in the past five years, infection in the right upper lobe with abscess, left lower lobe, and now left upper lobe (Fig. *7A*, *B*, *C*, and *D*). Because of the widespread condition and the fact that a bronchogram showed cylindrical bronchiectasis in all lobes, surgery was not advised. It was considered that we were dealing with mucoid impaction of the bronchi with bronchiectasis and abscess formation.

DISCUSSION

This condition must be suspected when pulmonary disease occurs in asthmatics and individuals having chronic obstructive bronchitis. The presence of the mucoid impaction causes a febrile episode. The fever may be initially high but responds well to chemotherapy and then persists as a low-grade daily temperature elevation. The associated cough is not severe. It may be a dry cough or productive of small amounts of mucopurulent secretion. General lassitude and a slight localized pleuritic pain may be present. Hemoptyses occurred in three of ten patients in our series.

Adequate sputum studies should differentiate this condition from tuberculosis. Differentiation from a putrid abscess may be more difficult. The history of asthma, the milder clinical course, the nonputrid sputum, and the position of the abscess cavity within the lung usually will make this differentiation possible. The eosinophil count was elevated slightly to twelve and fourteen in two of the patients in this report, but in the others the count was in the normal range. It may be impossible to rule out new growth in the lung without doing a thoracotomy. The surgeon, in operating upon such patients, must be on guard and must establish the true diagnosis before becoming committed to a too radical resection of pulmonary tissue.

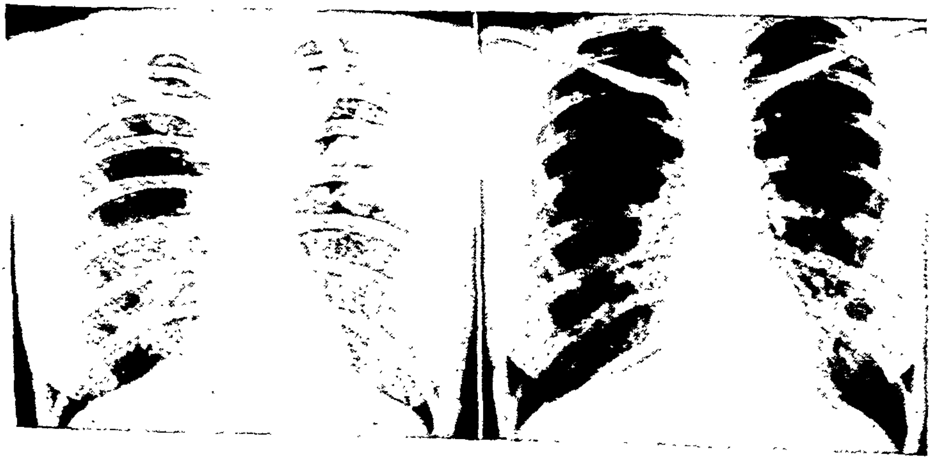
The diagnosis depends upon a careful history, laboratory studies, interpretation of roentgen films, and an awareness that the condition does exist in patients with an underlying asthmatic diathasis. Bronchoscopy is of little diagnostic aid because of the inaccessibility of the impaction. Occasionally, the diagnosis can be made with certainty when the patient gives a history of having coughed up a large, hard mucous plug.



A.

B.

Fig. 7. A and B.—(Case 10) Soft rounded shadow in upper pole of right hilum. Two months later a large abscess has formed beyond the blocked bronchus. Note new hilar shadows in left lung.



C.

D.

Fig. 7. C and D.—Three months later right upper lobe now clear. Dense shadows have appeared in left upper lobe. Eleven months later lung fields appeared clear.

NONSURGICAL MANAGEMENT

Where a definite diagnosis of mucoid impaction of the bronchus can be made, an attempt should be made to relieve the patient of this condition by nonsurgical measures. These include the use of chemotherapeutic agents, expectorant cough mixtures, and bronchodilators. Bronchoscopy is usually not helpful because of the inaccessability of the mucoid plugs. It has been suggested by some radiologists that roentgenogram therapy over a wide port in relatively small dosage acts as an expectorant and will aid these patients to rid themselves of the mucous plugs, thus improving bronchial drainage. We have had no experience with this type of therapy. ACTH also may be of benefit to these patients.

INDICATIONS FOR SURGERY

Indications for surgery in this condition are (1) persistence of suppuration with destruction of pulmonary tissue beyond impactions of mucus, (2) repeated hemoptyses from the associated cystic bronchiectasis, (3) the necessity of ruling out neoplasm when diagnosis is not certain.

This condition in its acute phase may appear to be life threatening, and yet can clear quickly leaving little demonstrable damage to pulmonary tissue. Due to the underlying asthmatic diathasis, these episodes may repeat in other segments of the lungs so that resection of pulmonary tissue must be limited to the actual lesion to preserve respiratory function. At the time of exploration the bronchial wall itself may have to be exposed to rule out neoplasm. With mucoid impaction the tumor is entirely within the bronchus. The bronchial wall is reddish brown in appearance and is edematous and partially necrotic. In certain instances, it will be necessary to resect an entire lobe rather than a segment so that the section through the bronchus can be made through normal tissue.

In certain individuals having this condition, surgery either is contraindicated or should be advised only because of compelling reasons. In a patient who has been followed over a period of years where multiple lesions have appeared and disappeared in segments of all lobes it would seem poor wisdom to attack a segmental atelectasis or suppuration beyond a plugged bronchus at any particular time. This patient has undoubtedly suffered grave damage to the pulmonary reserve and can ill afford to lose pulmonary tissue.

PROGNOSIS

The prognosis of this complication of asthma is the prognosis of the underlying disease. One can satisfactorily eradicate by resection a destroyed segment or lobe thus eliminating the symptoms of the complication. This does not insure that the patient will not repeat this same process in the future. In four of the ten patients that we have observed there have been recurrences of pneumonitis in other segments of the lung that were undoubtedly due to mucoid impaction of the bronchi.

SUMMARY

1. Mucoid impaction of the bronchi is a complication of asthma or chronic obstructive bronchitis.
2. The etiology and pathology of this clinical entity is discussed.
3. The roentgen appearance of the shadows cast by this disease may simulate tuberculosis, abscess and neoplasm.
4. Cystic bronchiectasis and fibroid pneumonitis are residual sequelae to this bronchial obstruction caused by mucoid impaction.
5. Surgery may be indicated to rule out neoplasm and/or to remove segments of lung destroyed by suppuration beyond the impaction.
6. Ten patients having this condition are reported. Eight had pulmonary resections. Two are being observed on nonsurgical management.

DISCUSSION

DR. LYMAN A. BREWER, III, Los Angeles.—The Association is indebted to Dr. Shaw and Dr. Paulson for bringing this condition to our attention in such a striking fashion. We have been interested in this problem for some time. The most important etiologic factors are: (1) an abnormal source of mucous secretion in the bronchial tree, and (2) conditions favoring retention of this mucus. Retained mucus may have an important effect on the spread and persistence of infection in other inflammatory conditions, particularly pulmonary tuberculosis. It will be well for us to consider various inflammatory diseases from this point of view.

I should like to comment briefly on only one phase of this problem, the persistence of mucous plugs in the bronchus as an important factor in producing pneumonitis and atelectasis in bronchiectasis. Here you have an abnormal source of mucus which, on occasion, is retained. In studying our resected pulmonary specimens, for bronchiectasis, we have found that lobar, segmental, and subsegmental atelectasis may be secondary to mucous blockage of the bronchus. In most instances this finding is not of great clinical importance; however, a most difficult problem is presented in the patients of the older age group who do not present histories typical of bronchiectasis and who have findings of bronchial obstruction that are impossible to differentiate from those of bronchiogenic carcinoma. The following case shows the difficulties that are encountered.

(Slide.) The patient was a 65-year-old waitress, complaining of severe hemoptysis of two weeks' duration. Roentgen examination of the chest revealed infiltration and pneumonitis with a possible central rarefaction in the lower right lung field. These findings pointed to an acute pulmonary abscess, possibly on the basis of bronchiogenic carcinoma. There was a history, on further questioning, that she had had a slight cough, productive of rather small amounts of mucoid sputum for a number of years, but no previous severe pneumonia. There was no evidence, as in the cases of Dr. Shaw and Dr. Paulson, of definite asthmatic bronchitis or bronchial asthma. She was put on intensive antibiotic therapy.

(Slide.) Two weeks later the roentgenogram showed a slight clearing of the lesion in the right lung field. The lateral film depicts atelectasis of the middle lobe. Antibiotics were continued, and she was symptomatically improved.

(Slide.) Bronchoscopy revealed no obstruction of the orifice of the right middle lobe. Purulent exudate issued from the right middle and lower lobe orifices. The roentgenogram demonstrated an enlargement of the shadow occupying the right middle lobe, similar to the cases presented by the speaker. Exploratory thoracotomy was advised. However, it was not until she brought up some foul sputum that consent to operation was obtained.

(Slide.) At exploration, the middle lobe was enlarged, boggy, and consolidated. Following removal of this lobe, cut section demonstrated a mucous plug which occurred exactly at the bifurcation of the two segments of the middle lobe, and thus could *not* be seen on bronchoscopy. On examination of the mucous plug, we could *not see* the laminations that the authors describe. It was degenerated mucus, with a good many fibrin strands, and bacteria were present which were apparently saprophitic. The chronic pneumonitis, suppuration, and lung abscess formation were present beyond the bronchial obstruction. In this case the bronchiectasis was the source of the sputum which became inspissated and obstructed the segmental branches of the bronchi. Secondary pneumonitis and abscess formation resulted. In patients in the cancer age group, it is impossible to rule out bronchiogenic carcinoma and, contrary to the cases in younger persons presented by the authors, surgical exploration is very definitely indicated.

DR. JEROME HEAD, Chicago.—This seems to me an original and excellent paper. I have never knowingly encountered this entity, but I am sure this has been due to a failure in observation. The subject interests me greatly because I am greatly interested in the sequellae of segmental and subsegmental bronchi. I would like to bring up the question as to why this entity does not occur more often. We know that plugs of mucus can and frequently do plug main bronchi, in which case there is atelectasis of a lobe. If plugs of mucus obstruct main bronchi occasionally it is reasonable to believe they obstruct segmental bronchi more frequently and finer bronchi very frequently.

In spite of this it is quite obvious that we do not encounter segmental atelectasis or subsegmental atelectasis. This can be explained only on the basis that there is a collateral air cirulation between lung segments and lung lobules. If it were not for this collateral circulation it seems certain that every bronchitis, by plugging the finer bronchi, would become a suffocating bronchitis. Without this collateral circulation there would develop a spreading patchy atelectasis from obstruction of small bronchioles. Because no air would get in behind these plugs to blast them out, the patient would eventually suffocate from a progressive patchy atelectasis. This of course does not happen. If it were not for collateral circulation, lobular atelectasis would be infinitely more frequent than lobar atelectasis and would be as difficult to relieve. Lobar atelectasis of course is relieved only when one interferes actively to get rid of the plug of mucus. This active intervention is necessary because no air can get in behind the plug to blast it free. I believe, therefore, that we should not speak of segmental or lobular or patchy atelectasis. When the parenchyma distal to an obstructed segment or lobule becomes opaque it becomes opaque because of infection and not primarily because of airlessness. To go further, we must ask ourselves what happens to an obstructed segment or lobule when infection does not occur. I believe we will find in these lung areas the development of the opposite of an atelectasis, in other words an emphysema. This is based on the assumption that the air enters the segment through the collateral circulation more easily than it passes back through the same channels. In this case the effect of continued coughing is to extend and stretch the obstructed segment. That this does occur is suggested by the following observations:

At the present time we have under observation a man in whom a single segment of the upper lobe which has been obstructed by a calcified gland has dilated to occupy the upper half of the thorax crowding the rest of the upper lobe and the lower lobe into the lower thorax. In another case in which there appeared to be a bleb occupying the lower half of the thorax operation revealed a tremendously emphysematous accessory lobe. This was connected to the lower lobe by a pedicle of lung tissue. There was no direct bronchial communication. In this case the air had passed into the accessory lobe through collateral alveolar channels. Apparently it was entrapped there and the result was a vesicular emphysema.

If we bear in mind the possibility that obstruction of a segmental or subsegmental bronchus without infection causes emphysema rather than atelectasis I believe that we will be on the way to recognizing an important clinical entity.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—This is an extremely important subject, particularly from the point of view of differential diagnosis in carcinoma, inflammatory lesions, cysts, and bronchostenosis. Also, in some cases of severe asthma a mucous plug may act as a trigger mechanism to set off bronchospasm. If such a condition can be found and eliminated by excision further attacks of asthma may disappear.

It was not quite clear to me, from what has been said, whether or not the mucous plug in the reported cases was the primary or secondary factor. In instances of this kind that we have observed there has been some condition which caused bronchial occlusion. The plug seemed to have resulted from concentration of mucus distal to the point of obstruction, therefore making it a consequence of another factor.

I should like to ask Dr. Shaw if they were not able to find in the pathologic specimens evidence of pressure from a gland, stenosis, or other organic change that might explain why the mucous plug developed.

DR. DEAN B. COLE, Richmond, Va.—Dr. Shaw has presented the problem of mucoid impaction beautifully but, as he has indicated, it is not new. The treatment of this condition rarely, if ever, is surgical. We have done lung mappings on many asthmatics in whom we have encountered bronchial spasm and mucoid impaction of bronchi. We have been able to demonstrate a reversible bronchiectasis in one such instance.

We have seen Loeffler's syndrome a number of times, and occasionally this will move from one lobe to another until eventually all lobes have been involved.

DR. SHAW (closing).—I wish to thank Dr. Brewer, Dr. Head, Dr. Overholt, and Dr. Cole for their discussion. In answer to Dr. Overholt's question I cannot be certain what is the actual underlying cause. I think the major cause is the thick tenacious type of mucus that these particular individuals have. In other words, it is similar to the child with chronic pancreatitis associated with bronchiectasis. Why the first accumulation occurs I do not know. Probably some thick material gathers in one bronchus, and it is my feeling that the accumulation from one bronchus, causing pressure obstruction on the neighboring branch bronchus, causes the formation of these plugs which is rather typical of this condition. In none of our patients have we found any significant glandular obstruction, but of course we are seeing them after suppuration has taken place, late in the process rather than when it began.

I did not mean to suggest, as Dr. Cole pointed out, that this is anything new. I did search the literature for a period of ten years and could find no direct reference to this particular entity. There are many reports describing atelectasis in asthma and Loeffler's syndrome. So far as I can tell no one has actually described the pathology of Loeffler's syndrome. They merely described the patchy, evanescent shadows that appear in the roentgenograms of people having this condition. It is my main concern to differentiate mucoid impaction from other diseases which have an entirely different prognosis. I feel that in some of these cases we will be forced to operate in order to rule out a disease that has a more severe prognosis and requires another form of treatment.

I also feel that in one of our cases, as Dr. Cole described, and as shown in the main body of the paper, we have had shadows of the same sort appear in every lobe of the lung, and the bronchogram demonstrates bronchiectasis in every lobe. This patient is not, however, in a good respiratory state as Dr. Cole suggests. She does have definite dyspnea, and I think she has suffered irreparable damage to the pulmonary bed. I certainly would not suggest that anyone should remove a segment of her lung at any particular time impaction should occur and a segmental atelectasis be found.

THE TREATMENT OF CARDIOSPASM: ANALYSIS OF A TWELVE-YEAR EXPERIENCE

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THE experience of the Mayo Clinic in the management of cardiospasm has been the subject of several previous reports from this institution. In 1906, Henry S. Plummer[1] recorded the first eleven cases of cardiospasm treated by him at the clinic, and he described a technique of dilatation which with relatively little modification has been employed in the great majority of approximately 1,800 patients with cardiospasm treated endoscopically at the clinic. In subsequent reports, Plummer[2, 3] reviewed his ever-increasing experience in the management of cardiospasm and his methods were either adopted or modified by other authorities interested in the treatment of this condition. In 1921, Plummer and Vinson[4] reported 301 cases, and the accumulated experience has since been described, in subsequent articles, by Vinson[5] and by Moersch.[6-8] Various problems in the diagnosis and management of cardiospasm have been the subjects of communications by other members of the staff[9-11]; however, there has been no complete review of the accumulated experience in the management of the cases of cardiospasm at the clinic since the report of Moersch[8] in 1936.

It is the principal purpose of this paper to describe and evaluate the results of treatment in 601 consecutive cases of cardiospasm in which dilatation was employed in the twelve years between Jan. 1, 1935, and Jan. 1, 1947. In addition, our experience with all cases of cardiospasm treated surgically at the clinic is reviewed. The etiologic and diagnostic aspects of cardiospasm will be reported in more detail in other communications.

DEFINITION OF CARDIOSPASM

Cardiospasm is a nonorganic stenosis of the lowermost portion of the esophagus. The portion involved by this closure is at, and immediately above, the juncture of the esophagus and stomach and is seldom more than 4 to 6 cm. long. Synonyms for cardiospasm include achalasia, mega-esophagus, idiopathic or diffuse dilatation of the esophagus and preventriculosis. All refer to obstruction at the site of the physiologic sphincter at the cardia (Fig. 1).

In order to evaluate the treatment of cardiospasm, it is most important that it be differentiated from other forms of esophageal spasm. The roentgenologic and clinical characteristics of diffuse spasm of the esophagus were described by Moersch and Camp[12] in 1934, and further elaborated upon by Schmidt[13] in 1939. This type of spastic constriction involves the smooth muscle portion of the esophageal musculature and may affect the lower third to two-thirds of the esophagus. The roentgenographic appearance of diffuse spasm

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varies considerably (Fig. 2). Hence, diffuse spasm of the esophagus is often referred to as pseudodiverticulosis,[14] localized spasm, segmental spasm, nonsphincteric spasm, reflex spasm, or even esophagitis. Many authorities have implied that diffuse spasm is an early stage of cardiospasm. In our experience, diffuse spasm is a clear-cut clinical entity not to be confused with cardiospasm, even though they may coexist occasionally. At times there may be some difficulty in distinguishing these conditions, both clinically and roentgenologically. However, the two entities may be readily differentiated by the passage of an olive-tipped bougie (41 F.) over a previously swallowed thread. In cardiospasm the obstruction is localized to a short segment of esophagus at the cardia. In diffuse spasm obstruction begins in the midesophagus and extends all the way to the stomach. The alteration in the roentgenoscopic appearance of the esophagus following dilatation of the cardia in cardiospasm should be mentioned. The activity of the esophageal musculature often is increased and the roentgenoscopic appearance may closely simulate that of diffuse spasm. This fact must be taken into consideration by the roentgenologist in his evaluation of conditions after treatment (Fig. 3).

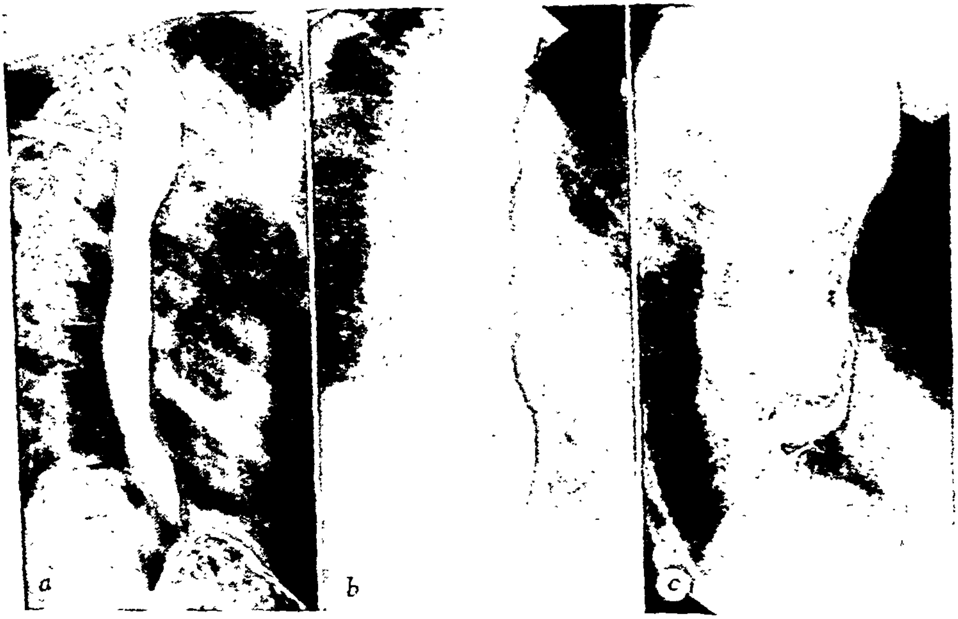

Fig. 1.—Cardiospasm. *a*, Moderate dilatation; *b*, diffuse dilatation; *c*, marked dilatation, angulation, and elongation.

Particular effort must be made to distinguish cardiospasm from other types of esophageal spasm. Diffuse spasm of the esophagus does not respond to treatment in the same manner as cardiospasm, and the hydrostatic dilator should not be used in cases of nonsphincteric spasm. Esophageal spasm which may be associated with other lesions of the esophagus or gastrointestinal tract, such as esophageal diverticula, hiatal hernia, peptic ulcer of the stomach or duodenum, and disease of the gallbladder also must be differentiated from cardiospasm.

TECHNIQUE OF DILATATION

Russel[15] described the first pneumatic dilator in 1898. Actually, Plummer's hydrostatic dilator was a modification of the Russel dilator adapted for water pressure and passed over a previously swallowed thread. The pneumatic principle was combined with Hurst's mercury-weighted tube by Tucker[16] and also by Browne and McHardy.[17] Some authorities continue to use dilators which are introduced through the esophagoscope.[18, 19]

The methods used at the clinic for dilating the cardia of patients with cardiospasm were described by Moersch[7] in 1933 and are essentially unchanged today. However, it is desirable to outline again the technique and procedure that we employ.

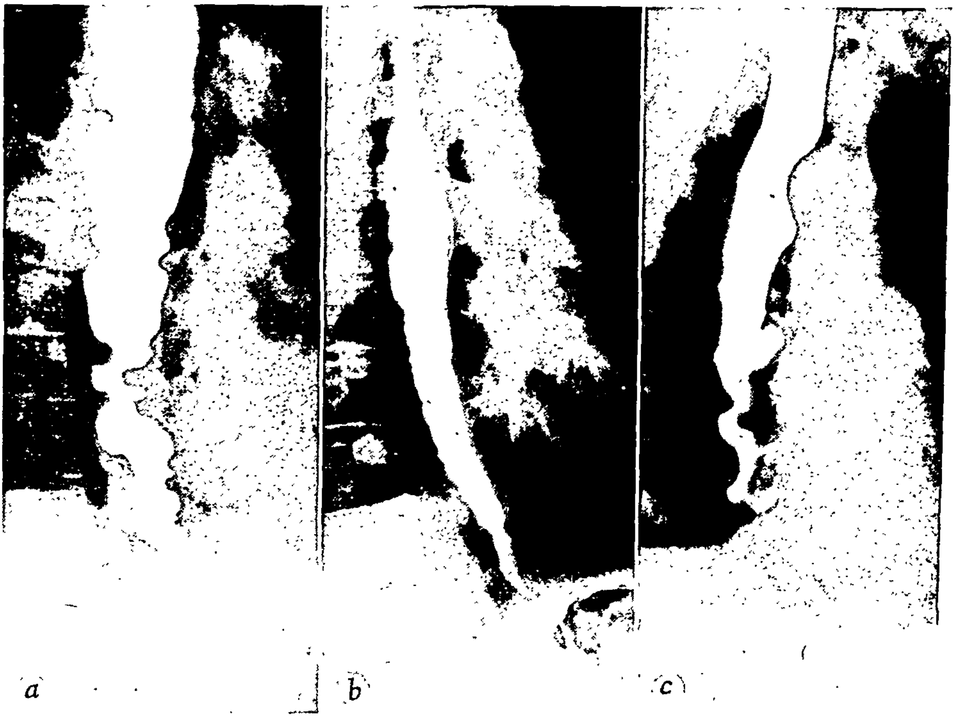

Fig. 2.—Diffuse spasm of esophagus. *a*, Diffuse irregular spasm; *b*, diffuse constant narrowing; *c*, pseudodiverticulosis.

Dilatations are carried out over a previously swallowed twisted silk thread. At present we are supplying our patients with "EE" buttonhole silk. The patient starts to swallow his thread twenty-four hours prior to the first dilatation; he swallows the thread slowly at the approximate rate of one foot (30.5 cm.) every hour and continues until fifteen feet (467 cm.) have been swallowed. He finds that eating food and swallowing liquids help him get the thread down, and he is permitted to eat all meals except the one prior to his treatment. During the night the thread hangs from the mouth, but it is usually advisable for him to place it between two teeth in order to avoid chewing it through during sleep. Treatment is not begun until the thread is completely anchored. Occasionally it is necessary to weight the end of the thread with lead shot. Nearly all patients are successful in getting a thread in place if they persist in their efforts, no matter how marked the obstruction.

Once the thread is firmly anchored, preliminary dilatations with metal sounds are carried out. The patient sits in a low chair facing the physician. A 41 F. olive-tipped metal bougie is attached to the end of a flexible whalebone staff, and this is passed over the thread into the stomach (Fig. 4, *a*). A second dilatation follows immediately, using a 50 or 60 F. Plummer sound guided over the thread by a flexible wire spiral (Fig. 4, *b*). Following the passage of these metal sounds, the patient rests for forty-eight to seventy-two hours. He is usually relieved of the greater part of his dysphagia and gets considerable food through the cardia during this period. The preliminary dilatation with sounds is important from a diagnostic standpoint, as the nature of the obstructing lesion can usually be determined by the "feel" of the sound as it passes through the cardia. Furthermore, subsequent hydrostatic dilatation is easier because esophageal retention has been relieved. Esophagitis improves rapidly following the initial dilatation and, in our opinion, the preliminary passage of sounds reduces the risk of the hydrostatic dilatation.

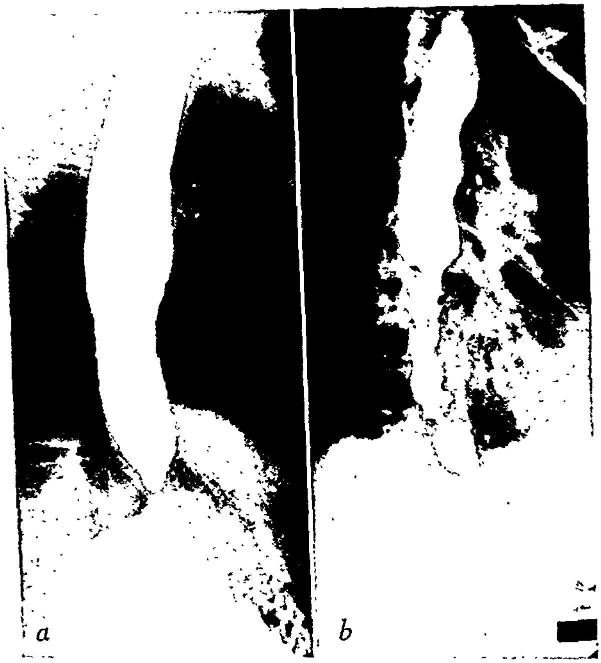

Fig. 3.—Effect of treatment on roentgenographic appearance of cardiospasm. *a*, Diffuse dilatation of esophagus before therapy; *b*, irregular and segmental spasm after hydrostatic dilatation.

When esophagoscopy is necessary for dilatation, the stenotic cardia must be located and a small flexible woven bougie, a stomach tube, or a small sound may be passed into the stomach. Then the esophagoscope itself may be passed into the stomach or a thread may be tied to the distal end of a bougie tube or sound, the esophagoscope withdrawn, and dilatations carried out over the thread.

The hydrostatic dilator has been described elsewhere.[7, 20] A flexible, whalebone staff is passed through the rubber tube, and a 41 F. olive-tipped metal bougie is attached to the threaded end of the whalebone staff (Fig. 4, *c*). The dilator is connected to the water source and pressure gauge and is tested before every treatment. This dilator is passed over the previously swallowed thread to insure the safety of the procedure, also to insure the proper placement of the bag in the cardia. The success of the procedure depends on exact placement of the bag before it is inflated. Just before passing the hydrostatic dilator,

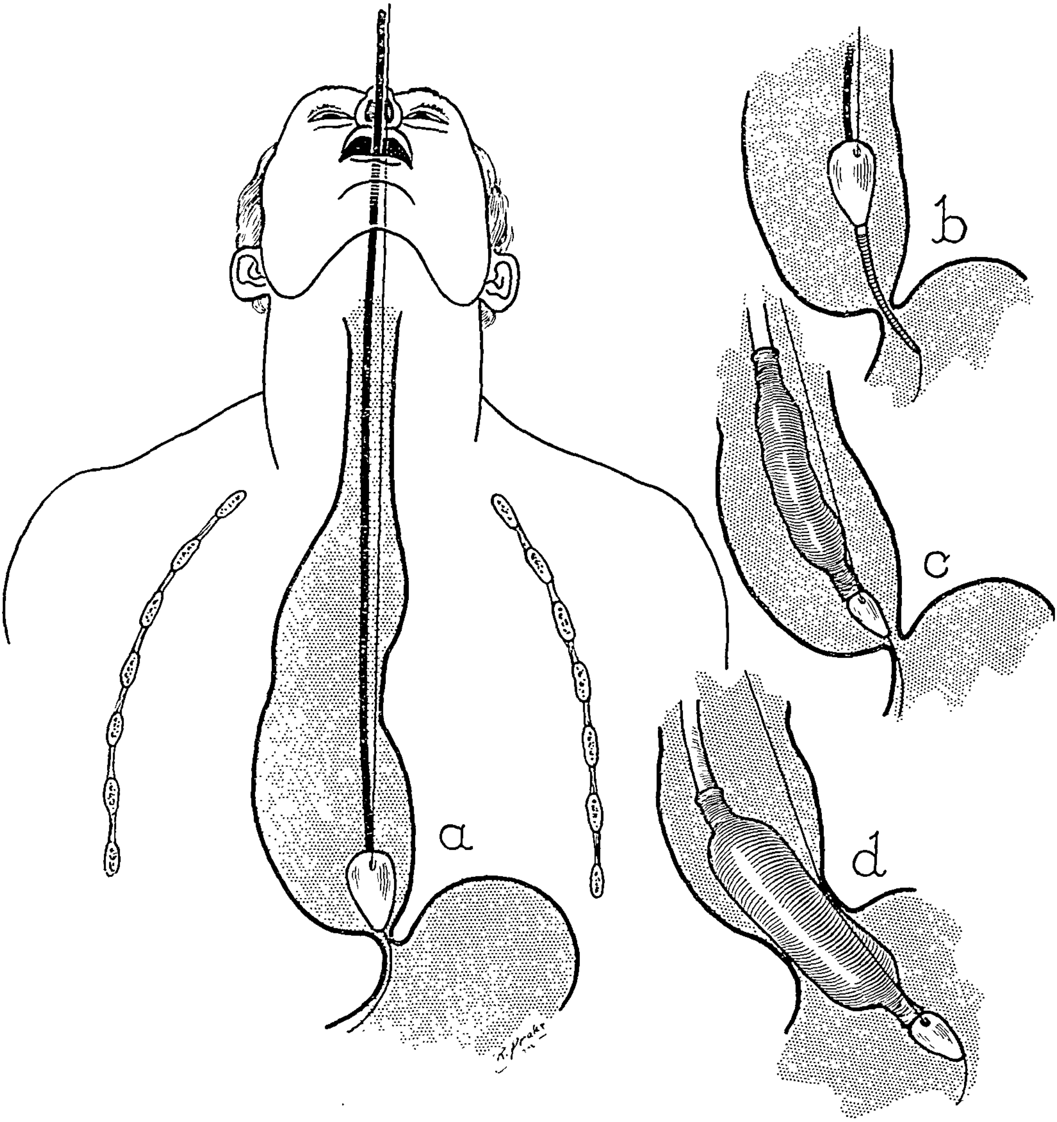

Fig. 4.—Method of performing dilatations. *a*, Passage of 41 F. olive-tipped bougie into the stomach; *b*, a 50 or 60 F. sound is passed into the stomach guided by a flexible wire spiral; *c*, the hydrostatic dilator is passed to the cardia; *d*, distention of the hydrostatic dilator across the cardia.

a 41 F. olive is passed into the stomach (Fig. 4, *a*). The exact distance from the point of the obstruction to the upper incisor teeth is determined and is marked off on the tube of the hydrostatic dilator. Thus the physician knows just how far to pass the hydrostatic bag. Once in place the bag is carefully inflated (Fig. 4, *d*) to 22 to 24 feet (9.5 to 10.3 pounds per square inch) of water pressure and the pressure maintained for several seconds. The patient usually will have considerable pain at the time of dilatation if the bag is properly placed. This pain subsides rapidly after the dilator is withdrawn. One evidence of a successful dilatation is the presence of a small amount of fresh blood on the distal end of the dilator.

The beneficial effects of the dilatation are immediate, and the patient spends the next two or three days testing his ability to swallow. He is advised to eat meat, apples, popcorn, and any other foods which may have given him particular difficulty. If complete relief from dysphagia is not obtained, the patient again swallows a thread, and a second hydrostatic dilatation is performed forty-eight or seventy-two hours after the first. It is seldom necessary to perform more than two or three hydrostatic dilatations, and the entire treatment program is usually completed in a week or ten days.

CASES STUDIED

We selected the period from Jan. 1, 1935, through Dec. 31, 1946, for our review of results of treatment of cardiospasm by hydrostatic dilatation for several reasons. Patients seen prior to Jan. 1, 1932, were covered in a previous report, and it was difficult to trace patients treated prior to 1935. We terminated our study at the end of 1946, so that the minimal period of follow-up would be four years. Some of the patients, therefore, have been under observation for sixteen years. Results based on a study of 601 patients, more than 75 per cent of whom have been successfully traced, should be statistically significant.

Not included in our series are sixty patients who were treated for cardiospasm during the twelve years, but who had also undergone dilatation at the clinic before 1935. Thus all our patients were new patients in the sense that we had not treated them for cardiospasm before. Of the 601 patients, 205 had had some type of dilatation therapy before we saw them.

For thirty-nine patients it was necessary to pass an esophagoscope to aid in the dilation. Forty-six patients were treated by passage of sounds without hydrostatic dilatations. The hydrostatic dilator was not used on some patients because of extreme youth, old age, or some associated serious disease. A few of these patients were so gratified by the temporary relief afforded by bouginage that they did not remain for hydrostatic dilatation. Four children, 6 years of age or less, had treatment by sounds only, and three obtained poor results. The fourth child has not been traced. Ten of the 46 patients (22 per cent) received satisfactory benefits from the passage of sounds alone. Vinson[20-21] and Carr[22] stated that only 10 per cent of patients treated with bougies alone received permanent relief.

In the period under consideration we treated 555 new patients with the hydrostatic dilator. For 452 of these patients, adequate follow-up studies were available and our conclusions concerning the method of hydrostatic dilatation are based on a study of these 452 patients.

RESULTS OF TREATMENT BY HYDROSTATIC DILATATION

We have set rather exacting criteria for satisfactory treatment. A good result from dilatation implies that lasting relief from dysphagia follows a course of treatment. In our study, results were classified as satisfactory or unsatisfactory. A satisfactory result means that the patient has had no dysphagia or only occasional or transient difficulty with swallowing since treatment. Patients in the satisfactory classification have remained virtually asymptomatic for four years or more* after treatment. A recurrence known to have developed in less than four years after treatment was classified as an unsatisfactory result. Also included as having unsatisfactory results were those individuals who reported partial relief of dysphagia but who still had significant symptoms (Table I).

We shall consider our experience in three categories: (1) results of the first course of treatment, (2) results of a second course of treatment for those patients who derived temporary relief of dysphagia after the initial course and who returned for further treatment, and (3) results of further treatment for patients who made three or more visits because of recurring dysphagia.

The Initial or First Course of Treatment.—Following preliminary passage of metal sounds, all of the 452 patients had one or more hydrostatic dilatations. The average number of hydrostatic dilatations was 1.67 per patient. The highest number for any individual patient at the initial visit was 12.

Of the 452 patients, 272 (60.2 per cent) were relieved of dysphagia for periods of four to sixteen years and are considered as having obtained satisfactory results. In the remaining 180, dysphagia recurred in less than four years, and the results were unsatisfactory according to our criteria.

Second Course of Treatment.—Ninety-four of the 180 patients who obtained an unsatisfactory result from the first course returned for a second course of treatment, and further hydrostatic dilatations were administered. Thirty-six of these 94 (38.3 per cent) obtained relief of dysphagia for four years or more and the results are classified as satisfactory. In fifty-eight cases, however, recurrence developed, and the results are considered unsatisfactory.

Three or More Courses of Treatment.—Twenty-six of the fifty-eight patients who obtained unsatisfactory results from a second course of treatment returned one or more times after the second course for further hydrostatic dilatations. Only five (19.2 per cent) of them were satisfactorily relieved by additional treatment.

TABLE I. *RESULTS OF TREATMENT BY HYDROSTATIC DILATATION OF 452 TRACED PATIENTS*

COURSE	PATIENTS TRACED	SATISFACTORY RESULTS		UNSATISFACTORY RESULTS	
		NO. OF PATIENTS	PER CENT	NO. OF PATIENTS	PER CENT
1	452	272	60.2	180	39.8
2	94	36	38.3	58	61.7
3 or more	26	5	19.2	21	80.8

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*A few patients known to be asymptomatic for more than two years but lost to follow-up thereafter are included in the satisfactory grouping.

All Courses.—It is evident that patients who did not respond satisfactorily to the initial course of treatment were less likely to obtain good results following a second course (38.3 per cent) or from any number of additional courses of treatment (19.1 per cent). Hence, if hydrostatic dilatation does not produce lasting relief of dysphagia at the first or second course, the prospects for success from further dilatation are not bright.

If the total number of satisfactory results in Table I are added, we find that 313 (69.2 per cent) of our 452 traced patients obtained satisfactory and lasting results; however, we did not have the opportunity of treating a second time all of the patients who had unsatisfactory results after the first course, nor did we treat all of those who failed to obtain satisfactory results from a second course. Had we been able to treat all of these patients and if the same percentage of satisfactory results noted in Table I had been obtained, our total number of satisfactory results would have been 341 (75.4 per cent) for two courses of treatment, or 362 (80.1 per cent) for all courses. Hence, if given the opportunity, we could anticipate satisfactory results from hydrostatic dilatation in 80.1 per cent of cases.

Number of Dilatations.—An analysis of the number of dilatations required in each case was made. When only one or two hydrostatic dilatations were necessary at the initial visit, satisfactory results were obtained in 64 per cent of the cases. However, if three or more dilatations were required to relieve dysphagia, the results were satisfactory in only 38 per cent. The experience was similar when patients returned for subsequent courses of treatment. This is further evidence to indicate that a favorable result can be anticipated from a prompt response to dilatation. If multiple treatments are required, the outlook is not as good.

In a large number of patients with cardiospasm there are many variable factors that must be considered in an evaluation of therapy. We have made an attempt to correlate some of these factors with the success of treatment.

Relation to Age.—Patients of all ages were treated, and, except for the very young, results were comparable in all age groups.

Fourteen patients treated by hydrostatic dilatation in this group were 15 years of age or less. Ten of these children were 11 to 15 years of age. Seven of the eight children in this age group who were traced did well. Results thus were comparable to those for adults. However, for the four children who were less than 10 years of age, results were not good. Only one got a good or satisfactory result. Results of use of sounds in young children were mentioned previously.

Relation to Duration of Symptoms.—The relationship of the duration of symptoms to the success of treatment is indicated in Table II. The ratio of

TABLE II. CORRELATION BETWEEN DURATION OF SYMPTOMS AND SUCCESS OF THERAPY OF 452 TRACED PATIENTS

DURATION OF SYMPTOMS (YEARS)	PATIENTS TRACED	SATISFACTORY RESULTS	
		PATIENTS	PER CENT
0-1	78	53	68.0
1-5	212	150	70.8
6-10	83	58	69.9
More than 10	79	52	65.8

satisfactory to unsatisfactory results is remarkably constant, regardless of the duration of the disease.

Relation to Size and Shape of the Esophagus.—The relationship between the size of the esophagus and the result of treatment is shown in Table III. Results were somewhat less favorable in the slightly enlarged and markedly enlarged organs (Fig. 1, *c*) than in the moderately and diffusely enlarged organs (Fig. 1, *a* and *b*). It is interesting that the slightly and markedly dilated esophagi responded less well to surgical treatment. It should be pointed out that in some cases of cardiospasm in which marked enlargement of the esophagus was present dilatation was impossible, and such cases have not been included here. In the surgical group there were eight such cases. Had these eight patients been taken into consideration in Table III, our results in the cases of marked enlargement of esophagus doubtless would have been less satisfactory than those presented. Our percentage of satisfactory results would have been about 58 per cent.

TABLE III. CORRELATION BETWEEN SIZE OF ESOPHAGUS AND SUCCESS OF TREATMENT OF 452 TRACED PATIENTS

DEGREE OF ENLARGEMENT OF ESOPHAGUS	TRACED PATIENTS	SATISFACTORY RESULTS	
		NUMBER	PER CENT
Slight	55	31	56.4
Moderate	200	139	69.5
Diffuse	127	98	77.2
Marked with torsion and angulation	70	45	64.3

A reduction in size of the esophagus may be anticipated in some cases after hydrostatic dilatation, although this diminution in size is uncommon in the markedly dilated esophagus. Actually, it is hard to predict with any certainty the likelihood of a decrease in size after treatment.

Relation to Type and Severity of Symptoms.—Obviously, results of treatment by dilatation were most dramatic in those patients who had marked obstruction and who regurgitated food most readily.

The relationship of pain to cardiospasm will be considered in another communication. In sharp contrast to figures given by some authors, pain occurred in less than 30 per cent of our cases. When pain was a prominent symptom of cardiospasm, we frequently obtained a less satisfactory result. Of 132 patients who complained of pain, only 51.5 per cent obtained satisfactory results, as opposed to 69.2 per cent for the whole group. In a number of cases our treatment was effective in relieving the obstruction and regurgitation of cardiospasm, but the patient continued to have pain.

Pain is a much commoner symptom in the syndrome of diffuse spasm of the esophagus than it is in cardiospasm. Although pain may be a feature of cardiospasm, an unusual amount of pain should make one wonder whether some other esophageal disease is associated.

Relation to Associated Disease.—Certain associated disease processes had an influence on the result of therapy. Patients who had associated pulsion diverticula of the upper or lower part of the esophagus were more difficult to treat, and results were poor in this group. Patients who had hiatal hernia in association with cardiospasm were likewise less favorable candidates for treatment. We have learned that hiatal hernias of the short esophagus type are particularly troublesome and that dilatation of cardiospasm in such cases may be hazardous. The development of a short esophagus after rupture of the lower part of the esophagus has been noted. Fig. 5, *b* shows the condition after rupture of the lower part of the esophagus by the hydrostatic dilator. In retrospect there may have been a small hiatal hernia before dilatation (Fig. 5, *a*).

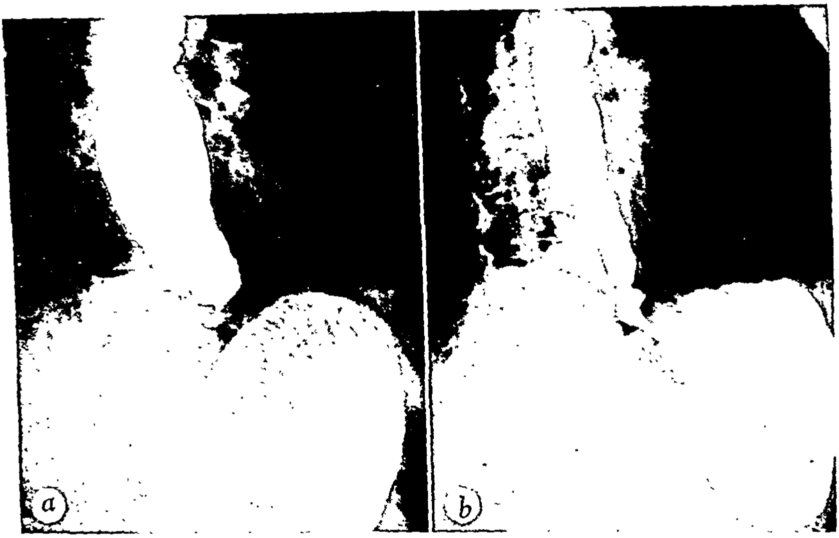


Fig. 5.—Development of short esophagus following rupture of lower part of esophagus by hydrostatic dilator. *a*, Cardiospasm before treatment; *b*, cardiospasm with small hiatal hernia.

In forty of our 601 cases, diffuse spasm of the esophagus was associated with cardiospasm. The diagnosis of the combined conditions can be suspected roentgenographically and confirmed by the findings at dilatation. When both conditions are present, results of treatment are difficult to evaluate. The obstruction of cardiospasm will often be relieved while the pain of diffuse spasm persists. The presence of associated diffuse spasm probably accounts for the less favorable results noted (Table III) in those patients with slight enlargement of the esophagus. The same considerations apply to surgical results (as will be evident in Case 3).

Painful Reactions.—As previously stated, most patients experience pain in the subxyphoid area during hydrostatic dilatation. This usually subsides rapidly after removal of the dilator. In twenty-one of our cases rather severe epigastric or substernal pain developed several hours after hydrostatic dilatation. This pain was often referred to the neck, ears, shoulders, or arms. None of these patients showed any objective evidence of esophageal perforation, nor were there any physical findings of significance. Some of them did have some fever, and several were kept in the hospital for observation. In none of these cases were there any serious consequences. In our opinion, these painful reactions were the result of spasm of the esophagus secondary to the trauma of the procedure. Results of therapy in this group were comparable to those of the entire series.

COMPLICATIONS OF HYDROSTATIC DILATATION

The forceful dilatation of the cardia to a diameter of 3 to 4 cm. by a set of silk and rubber bags distended with water carries a certain risk. It is inevitable that a number of patients will experience undesirable symptoms or complications as the result of the treatment (Table IV).

TABLE IV. COMPLICATIONS FOLLOWING HYDROSTATIC DILATATION OF 555 CASES

TYPE OF COMPLICATION	CASES	MORTALITY
Esophageal "split"	10	2 (0.36 per cent)
Aspiration pneumonitis	6	0
Hematemesis	7	0

Hemorrhage.—Only seven patients had significant bleeding after hydrostatic dilatation. A small amount of blood on the hydrostatic dilator is expected after such dilatations, but serious bleeding is unusual. When hematemesis or melena occurs after dilatation, some associated process should be looked for, such as gastric ulcer, hiatal hernia, or associated stricture. Two of our patients who bled are known to have had short esophagus, and one patient had a gastric ulcer. One of these patients subsequently underwent esophagogastrostomy elsewhere with good results.

Aspiration Pneumonitis.—Aspiration pneumonitis developed after hydrostatic dilatation in six of our cases. All of these patients promptly recovered. It is our feeling that preliminary passage of sounds relieves esophageal retention and reduces the frequency of aspiration of esophageal content.

Rupture of the Esophageal Wall Near the Cardia.—This complication occurred in ten cases after hydrostatic dilatation. In such cases severe epigastric and substernal pain develops a few hours after dilatation. At the onset the pain is hard to distinguish from that of a painful reaction. However, almost invariably left pleuritic pain with both the physical signs and roentgenologic evidence of pleuritis appears. Effusion may develop and require thoracentesis.

Of these ten patients, two died of mediastinitis, one in 1937, and the other in 1940. In one case (1943), left empyema developed which required surgical drainage. This patient recovered but subsequently had a short esophagus type of hiatal hernia with stricture at the esophagogastric junction (Fig. 5, *a* and *b*). In another case a left transthoracic incision was made after the rupture, and the lower part of the esophagus and upper part of the stomach were resected. This patient did well, and is considered in the discussion of surgical management of cardiospasm. The remaining six patients made complete recoveries with conservative therapy.

The management of esophageal rupture was reviewed by Seybold, Johnson, and Leary.[23] When mediastinitis follows dilatation of the cardia, drainage of the posterior mediastinum is the safest procedure. When prompt surgical drainage is combined with adequate antibiotic therapy, the mortality from hydrostatic dilatation should be negligible. Our two fatalities occurred prior to the era of penicillin and other effective antibiotics and before radical surgical treatment was considered feasible.

COMMENT ON TREATMENT BY HYDROSTATIC DILATATION

It is often stated that successful management of cardiospasm by the method of dilatation requires that treatments be administered at frequent intervals over long or indefinite periods of time. In our experience, this has not been the case.

The majority of patients who have cardiospasm can be successfully treated by hydrostatic dilatation. Not only are the immediate results satisfactory, but these patients have been relieved for periods ranging from four to sixteen years by one or two courses of treatment. It is also obvious that relief of dysphagia is usually accomplished by one or two hydrostatic dilatations. If permanent relief is not obtained during the first or second course of treatment, further dilatations are less likely to produce a favorable and lasting result.

Our studies show that approximately 20 per cent of patients cannot be expected to obtain permanent benefit by hydrostatic dilatations. A good many of these patients are improved but still have dysphagia; others have to return at intervals for repeated dilatations in order to control their symptoms. Although we do not consider the results satisfactory in these patients, the periodic dilatations enable them to live comfortable lives.

On the whole, the results of treatment in the present series of patients are comparable to those reported by Moersch.[7, 8] The experience of Nagel,[24] using methods similar to ours, is also comparable. In a group of fifty-seven patients, he obtained good results from dilatation in 73 per cent. Nagel also emphasized the point that a single forceful dilatation produces much better results than repeated "half-hearted attempts." Freeman[25] also favored the use of the hydrostatic dilator and apparently has had good results with one dilatation.

Successful dilatation therapy after the proper diagnosis of cardiospasm depends on two factors: (1) accurate placement of the dilator in the cardia, and (2) severe forceful dilatation of the physiologic sphincter by an expanding dilator. Obviously, the passage of simple bougies or mercury-weighted tubes does not often produce sufficient expansile force at the cardia to have any lasting effect. In most cases such procedures must be repeated frequently. In our opinion, the previously swallowed thread permits accurate placement of the dilator and greatly adds to the safety of the procedure. In each case the dilatation, however forceful, must be administered at the level of the constriction if favorable results are to be anticipated.

In a recent communication, Sifers and Crile[26] reported that only eleven of ninety-two patients treated by means of dilatation obtained satisfactory results; sixteen of their patients were considered improved, forty slightly improved, and eighteen unimproved. Their dilatations were performed with Hurst mercury bougies and the Browne McHardy pneumatic dilators. We must conclude that effective dilatation was not obtained in most of their cases. Similarly, Fritz, Clark, and Adams[27] advocated surgical treatment because of the "temporary results usually obtained by mechanical *dilatation* from above." Their temporary results do not conform to our experience.

We realize that the method of hydrostatic dilatation employed by us is a drastic procedure. There is nothing "half hearted" about it, and the procedure carries risk. Ochsner, DeBakey, and DeCamp[28] recently called attention to the 2.8 per cent mortality in the 804 cases reported by Moersch.[7] As previously stated, this was reduced to 0.36 per cent in the twelve-year period under consideration, and in the past eleven years there have been no deaths. We must anticipate a 3 per cent incidence of complications. However, with chemotherapy and prompt surgical intervention, mortality in the future should be negligible.

SURGICAL CONSIDERATIONS IN THE TREATMENT OF CARDIOSPASM

We believe that operative treatment should be considered in all cases in which hydrostatic dilatations or more conservative measures do not give satisfactory results. From this study it appears that about 20 per cent of patients will not respond to conservative methods of treatment such as dilatation. It is difficult to anticipate which patients will fail to respond to conservative measures, but in general those who have marked enlargement of the esophagus and retention are most likely not to respond.

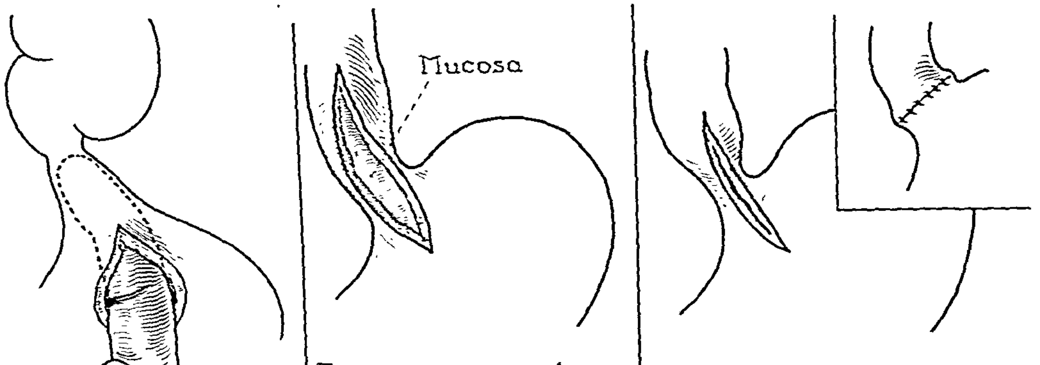
Several of the operative procedures such as vagotomy, sympathectomy, and phrenicectomy are based on the neurologic concept of the etiology of cardiospasm. These operative procedures have given some satisfactory results but none have given uniform and consistently good results. One of the earlier surgical procedures was that advocated by von Mikulicz[29] of transgastric retrograde dilatation. This procedure gave only temporary relief and is now rarely, if ever, utilized.

The more common types of operative procedure utilized at present are directed to either short circuiting the obstruction or attacking the constricted area (Fig. 6). These procedures are as follows: (1) esophagogastrostomy as advocated by Heyrovsky[30] is a side-to-side anastomosis between the dilated portion of the esophagus and the fundus of the stomach; (2) cardioplasty as advocated by Wendel[31] consists of a longitudinal incision in the lower part of the esophagus and the cardiac end of the stomach through the region of obstruction. This incision is closed in a transverse direction to enlarge the opening between the lower end of the esophagus and stomach; (3) a combination esophagogastrostomy and cardioplasty was advocated by Gröndahl[32] in which an anastomosis is made between the lower part of the esophagus and cardiac end of the stomach after a U-shaped incision is made through the constricted region of the esophagus and cardia; (4) extramucous esophagocardiomyotomy as advocated by Heller[33] consists of an incision down to the mucous membrane in the longitudinal circulatory muscles in the region of the obstruction; (5) cardiectomy and resection of the lower part of the esophagus and esophagogastric anastomosis may be used.[34, 35] For the first four procedures the abdominal approach usually is used, and, for the last procedure, the transthoracic approach.

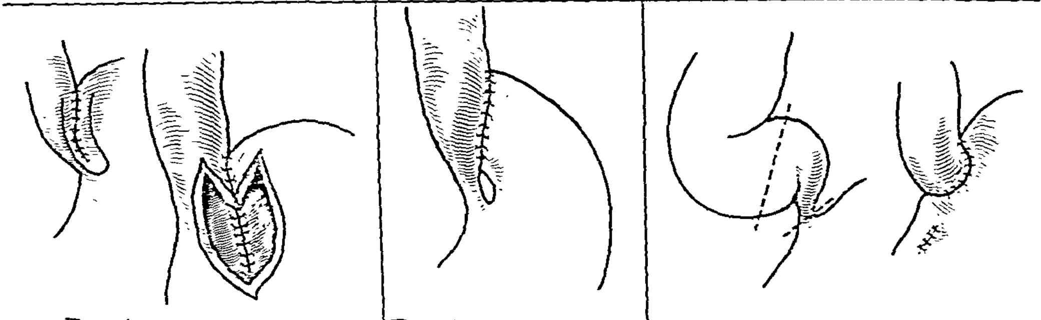
SURGICAL EXPERIENCE AT THE MAYO CLINIC

Twenty-three patients have been treated surgically at the clinic for cardiospasm, and most of the operative procedures just mentioned have been utilized.

One patient had two operations, making twenty-four operative procedures by several different surgeons (Fig. 6). Many of these twenty-three cases have been reported in the literature. Plummer[1] in 1906 reported a retrograde transgastric dilatation performed by W. J. Mayo in 1905; Judd, Vinson, and Greenlee[36] reported two cases in 1929; Craig, Moersch, and Vinson[37] reported one case in 1934; Gray and Skinner[38] reported seven cases in 1940, including the previously reported cases in which operation had been performed at the clinic up to that time. Clagett, Moersch, and Fischer[39] reported four cases in 1945.



Transgastric dilatation (retrograde) | Extramucous esophago-cardiomyotomy Zaaijer (Heller) | Cardioplasty Wendel



Esophagogastrostomy with cardiotomy Gröndahl | Esophagastrostomy (side to side) Heyrovsky | Resection of cardia and lower esophagus

Fig. 6.—Various types of operative procedures performed in this series of twenty-three patients.

The chief indication for surgical intervention in most of our twenty-three cases was an unsatisfactory result from dilatation or other conservative methods of treatment. In many of our cases, dilatation and thickening of the walls as well as elongation of the esophagus were present. These marked secondary changes are usually associated with the more severe types of cardiospasm. We believe that the results of surgical treatment are more likely to be unsatisfactory when these marked secondary changes are present. This, however, is only one of the factors associated with unsatisfactory results from operative treatment. There is increasing evidence to indicate that the type of operative procedure is important in the long-term results. Most procedures permit regurgitation of gastric secretions which cause inflammatory changes in the lower part of the esophagus.

The operative procedures utilized for our twenty-three patients are shown in Fig. 6. Four patients were treated by transgastric retrograde dilatation, one by sympathectomy* and one by both retrograde dilatation and sympathectomy. Of these six patients, five had poor results and one patient who had retrograde dilatation alone obtained a good result. The only operative death in the twenty-three cases followed transgastric retrograde dilatation. The patient died of pneumonia five weeks following the dilatation. These procedures have not been done in recent years.

In four of the twenty-three cases esophagogastrostomy of the Heyrovsky type was used. Results were poor in two cases and good in two. All four of these patients had marked dilatation of the esophagus before operation, and three had retention of food. Symptoms had been present for two to twenty-five years before operation. One patient was 26 years of age, and three were more than 60 years of age. Of the two patients who obtained poor results, one, aged 69 years, had had symptoms for twenty-five years and had been treated by twelve dilatations. This patient died four years after operation from hemorrhage resulting from esophagitis and ulceration. The other was 26 years of age and had had symptoms for two years. Operation relieved the symptoms for one year, and then symptoms of obstruction and esophagitis recurred.

In five cases, esophagogastrostomy of the Gröndahl type was employed. Results were poor in three cases, fair in one, and good in one case. The ages of these patients were from 20 to 57 years, and symptoms had been present for nine to twenty-four years. All five had enlargement of the esophagus, and three had angulation with retention. One patient who had a poor result was 31 years of age, and had had symptoms for nine years (Case 1). Another was relieved for three years, and then symptoms recurred. Six and one-half years after the first operation a second operation, transthoracic cardiectomy, was performed. Since the second operation four months ago, symptoms have been relieved. The third patient who obtained a poor result from this operation had a markedly enlarged and elongated esophagus with marked retention of food (Case 2).

In four of the twenty-three operative cases, cardioplasty of the Wendel type was carried out. Results were good in one case, fair in two, and poor in one. Of the last three patients, one had symptoms of esophagitis on examination six years after operation, one was having attacks of dysphagia six years following operation, and the third obtained no relief from the operation.

In two of the twenty-three cases, transthoracic resection of the lower part of the esophagus and cardia with esophagogastric anastomosis was carried out. In one case the operation was done four days after rupture of the esophagus following an esophagoscopic examination. Five and one-half years have now elapsed since the operation, and results are good. In the other case the operation was carried out six years after esophagogastrostomy of the Gröndahl type which did not relieve symptoms. As only four months have elapsed since the second operation it is too soon to evaluate the result, but thus far the patient has had a good result.

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*This case reported by Craig, Moersch, and Vinson[37] as a case of cardiospasm has been reclassified and is now considered a case of diffuse spasm of the esophagus.

In the remaining three of the complete group of twenty-three cases, esophagocardiomyotomy of the Heller type was used. Results were considered good in one case (Case 3). In the other two the operation was performed within the last eight months, and it is too soon to determine anything but the immediate result. This, however, has been good in both cases. A transthoracic approach was used in two cases, and an abdominal approach in one case.

CASE REPORTS

Four cases in which one of us (Harrington) performed the operations are reported.

CASE 1.—A man, aged 31 years, was first admitted to the clinic Jan. 25, 1942. For nine years symptoms of dysphagia, with substernal pain, regurgitation, and vomiting, as well as retention of food in the esophagus had become progressively more severe. He lost 15 pounds (6.8 kg.). Dilatation of the esophagus elsewhere had given some temporary relief.

Roentgenographic examination of the esophagus showed cardiospasm with obstruction at the cardia, marked enlargement and tortuosity of the esophagus, and retention of large amounts of food.

Hydrostatic dilatation was done in February, 1942, without relief of symptoms.

On February 21 esophagogastrostomy with cardiotomy (Gröndahl type) was performed. The patient had no difficulty in swallowing food after operation, but had a burning in the lower part of the esophagus which was relieved by alkalies. He continued to have burning pain low in the esophagus and some, but not severe, attacks of dysphagia. Esophagoscopic examination six months after operation showed esophagogastritis with ulceration low in the esophagus. The patient has returned several times since and has had two courses of dilatations without any improvement of symptoms.

Nine years after operation the patient wrote that his general health was good, he was working every day, but had more or less constant burning in the esophagus after meals which was relieved by alkalies. He also was having periodic attacks of dysphagia and vomiting. He said, "I am grateful for the operation as I get along a thousand times better than before the operation."

We, however, consider the result in this case poor. The improvement in subjective symptoms was due to the release of the obstruction by the operation but this type of operative procedure employed has permitted the gastric secretions to flow into the esophagus. The ensuing esophagitis is the chief cause of his present symptoms. However, some patients have obtained relief of symptoms from this type of operative procedure. It is possible, therefore, that surgical treatment might have been more effective if it had been done before the secondary changes due to dilatation and retention of food had developed in the esophagus.

CASE 2.—A woman, aged 45 years, was first admitted to the clinic Oct. 5, 1939, because of attacks of dysphagia, regurgitation, and vomiting of food. The number and severity of these attacks had been increasing, and at this time she was vomiting large quantities of food every night.

The roentgenogram showed marked dilatation and obstruction at the cardia and cardiospasm (Fig. 7, *a*). A satisfactory esophagoscopic examination could not be done until two and one-half liters of food and secretions were removed from the markedly dilated and tortuous esophagus. After repeated unsuccessful attempts, a thread was passed (through a stomach tube) through the obstruction at the cardia and the esophagus was dilated over the thread with a 60 F. sound. Repeated attempts to pass the esophagoscope over the thread through the cardia into the stomach were unsuccessful. Because it was impossible to treat the obstruction at the cardia by dilatation methods, surgical treatment was advised and refused at that time.

The patient returned Sept. 3, 1948, and stated that unsuccessful attempts had been made in her home locality to dilate the esophagus both from above and below. In 1940 gastrostomy had been done for feeding and when the opening was closed symptoms recurred. They had become progressively more severe and she had lost 50 pounds (22.7 kg.). Roentgenographic and esophagoscopic examinations showed a markedly enlarged tortuous esophagus with obstruction at the cardia. Four liters of foul material were aspirated from the esophagus. Oct. 18, 1948, esophagogastrostomy (Gröndahl type) with cardiotomy was done through an abdominal incision (Fig. 6).

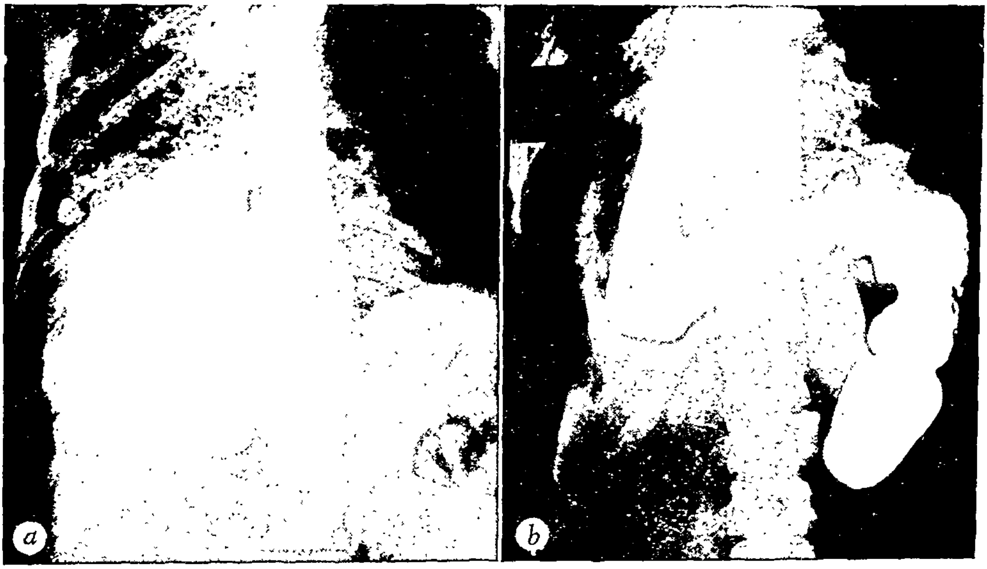

Fig. 7 (Case 2).—*a*, On admission cardiospasm with extreme dilatation and elongation of esophagus. Marked retention of food and secretions. Esophagus occupies most of right half of the thorax. *b*, Three months after abdominal esophagogastrostomy with cardiotomy (Gröndahl). Dilatation of esophagus is less marked.

After the operation the dysphagia and retention of material in the esophagus lessened (Fig. 7, *b*), but daily aspiration of the esophagus was continued. Improvement, however, was only temporary for retention of large quantities of food, pain below the sternum and to the right shoulder developed and severe hemorrhages occurred. Resection of the lower part of the esophagus and cardia of the stomach and vagus nerves was performed elsewhere in November, 1949.[27] A few weeks after this operation, she had massive hematemesis. In December, 1949, the antrum of the stomach was resected elsewhere. Convalescence was complicated and she died five months later.[27]

This case shows the serious complications that can arise from cardiospasm as well as the difficulty that may follow treatment after these complications have developed. We believe that when elongation and retention of secretions are present the danger of esophagitis complicating any type of operation which permits gastric juice to enter the esophagus is greater than in cases in which these changes have not occurred. The surgical problem in these cases is not only the removal of the obstruction, but also correction of the elongation of the esophagus which acts as a reservoir for food and secretions. These patients may be best treated surgically by resection of the elongated portion of the esophagus with esophagogastric anastomosis. However, it would be better to institute surgical treatment early in the course of the disease and thus avoid the necessity for these more extensive operative procedures.

CASE 3.—A woman, aged 30 years, came to the clinic Jan. 26, 1949. She had had intermittent and progressively increasing difficulty in swallowing since childhood. During the past year she had had attacks of severe dysphagia, vomiting, and spasm of the lower part of the esophagus as well as pain between her shoulder blades.

Esophagoscopic and roentgenographic examinations at the clinic showed marked dilatation of the upper third of the esophagus with a fluid level due to pouching of the wall of the esophagus but no opening suggestive of a diverticulum, and moderate dilatation of the lower two-thirds of the esophagus with narrowing of the cardia and some associated diffuse spasm. Hydrostatic dilatations were carried out from Feb. 9 to 17, 1949, without relief of symptoms.

The patient returned to the clinic in April, 1949, because her symptoms were more marked than before dilatation. Surgical treatment was advised. She was told that we could not promise complete relief of all of her symptoms inasmuch as they were due to diffuse spasm of the entire lower part of the esophagus as well as to cardiospasm. We thought relief of the obstruction at the cardia might lessen her symptoms.

On May 4, 1949, an extramucous esophagocardiomyotomy (Zaaijer modification of the Heller operation) was carried out with a left transthoracic approach.

The immediate result was entirely satisfactory in that dysphagia was relieved. Roentgenographic studies at the time of her dismissal revealed some spasm of the lower part of the esophagus. When she returned for further examination one year later, she had been completely relieved of the obstructive symptoms and was able to eat all types of food without difficulty. However she occasionally had attacks of mild spasm associated with some pain between her shoulder blades when emotionally upset or under stress. Two years after the operation (February, 1951) she wrote that her condition was essentially the same except for some gradual improvement.

Symptoms in this case were due to cardiospasm and diffuse spasm of the lower part of the esophagus. Extramucous esophagocardiomyotomy relieved the obstructive symptoms due to the cardiospasm but, as expected, some of the symptoms due to the spasm of the lower part of the esophagus have continued. This case exemplifies the importance of determining clinically whether the condition is cardiospasm or if there is also an associated spasm of the lower part of the esophagus as the operative treatment for cardiospasm will not correct the diffuse spasm of the lower part of the esophagus.

CASE 4.—A man, aged 23 years, first came to the clinic Nov. 9, 1945, because of regurgitation of food and coughing and choking at night. For approximately two years he had had episodes of obstruction on swallowing food associated with pain at the lower end of the sternum. These attacks had become more frequent and severe.

Roentgenograms of the esophagus and stomach revealed cardiospasm with moderate dilatation of the esophagus. One dilatation completely relieved him for two months. Then his previous symptoms gradually reappeared and became more severe.

He returned to the clinic in April, 1947. The roentgenographic appearance of the esophagus was essentially the same as at the previous visit. Hydrostatic dilatations of the esophagus on April 14, 1947, relieved his symptoms for nearly a year.

He returned again Feb. 22, 1951. The roentgenogram taken at this time showed slight dilatation of the esophagus (Fig. 8, *a*). Esophagoscopic examination showed a moderate degree of esophagitis in the lower half of the esophagus but no ulceration. On completion of his examination, operative treatment was advised because he had received only temporary relief from the two previous dilatations, the second of which was a thorough hydrostatic procedure, and it was thought that further dilatations would not give more than temporary relief. Accordingly, on March 2, 1951, a Zaaijer modification of the Heller extramucous esophagocardiomyotomy was done with a left transthoracic approach.

The immediate result was satisfactory. Symptoms were relieved and the roentgenogram (Fig. 8, *B*) before dismissal revealed a normal esophagus with no obstruction at the cardia.

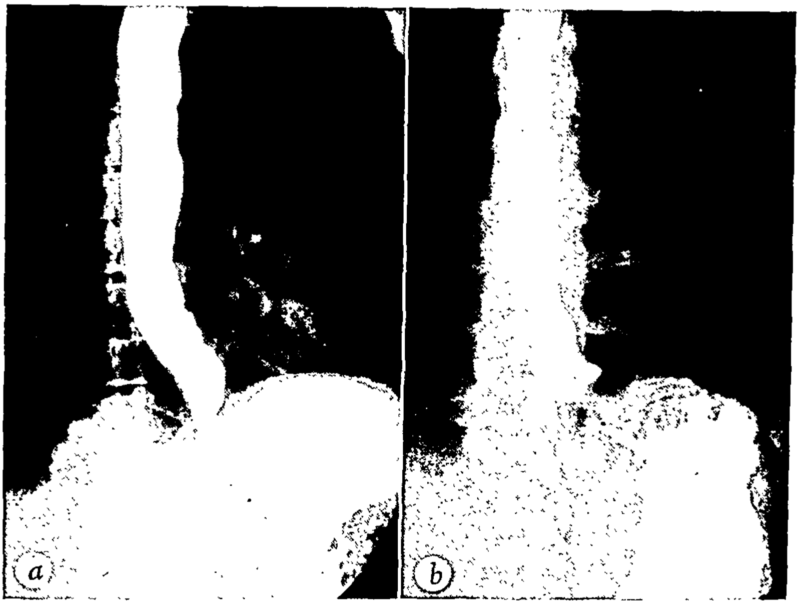

Fig. 8 (Case 4).—*a*, On third admission after two previous dilatations cardiospasm with some dilatation of esophagus; *b*, three weeks after extramucous esophagocardiomyotomy; no obstruction at cardia and esophagus is normal.

In this case surgical treatment was instituted early before the secondary changes and retention had occurred. We believe this is the best course to follow in treatment of cardiospasm which does not respond to dilatation. We are hopeful that the extramucous esophagocardiomyotomy will give complete relief. We hope, too, that it will prevent the esophagitis which so frequently follows a direct anastomosis between the esophagus and stomach when there is no sphincteric control to prevent the regurgitation of gastric secretions into the esophagus.

TECHNIQUE OF EXTRAMUCOUS ESOPHAGOCARDIOMYOTOMY EMPLOYED

The extramucous esophagocardiomyotomy of the Heller type employed in our cases is the modified procedure of Zaaijer,[40] which is advocated by Maingot[41] and others.[42-45] This technique and the transpleural approach used in the two last cases reported in this series will be described (Fig. 9).

A left posterolateral incision is made over the eighth rib on the left and the posterior two-thirds of the rib is resected. The pleural cavity is entered through the posterior periosteum of the eighth rib. The suspensory ligament of the lower lobe of the left lung is incised and the lung is retracted. The mediastinal pleura over the esophageal ring and the lower part of the esophagus is incised and the esophagus is elevated into the pleural cavity. The attachments of the diaphragmatico-esophageal membrane at the esophageal ring are separated from the lower part of the esophagus and cardia by blunt dissection. The esophageal ring is incised and the cardiac end of the stomach is brought into the pleural cavity. The entire constricted area of the lower part of the esophagus and cardia of the stomach are exposed and straightened out by traction. The vagus nerve is retracted to the inner side. An incision is made upward through the anterior surface of the longitudinal and circular muscles of the constricted portion of the esophagus well into the dilated portion of the esophagus and downward through the circular muscle of the cardia and muscle

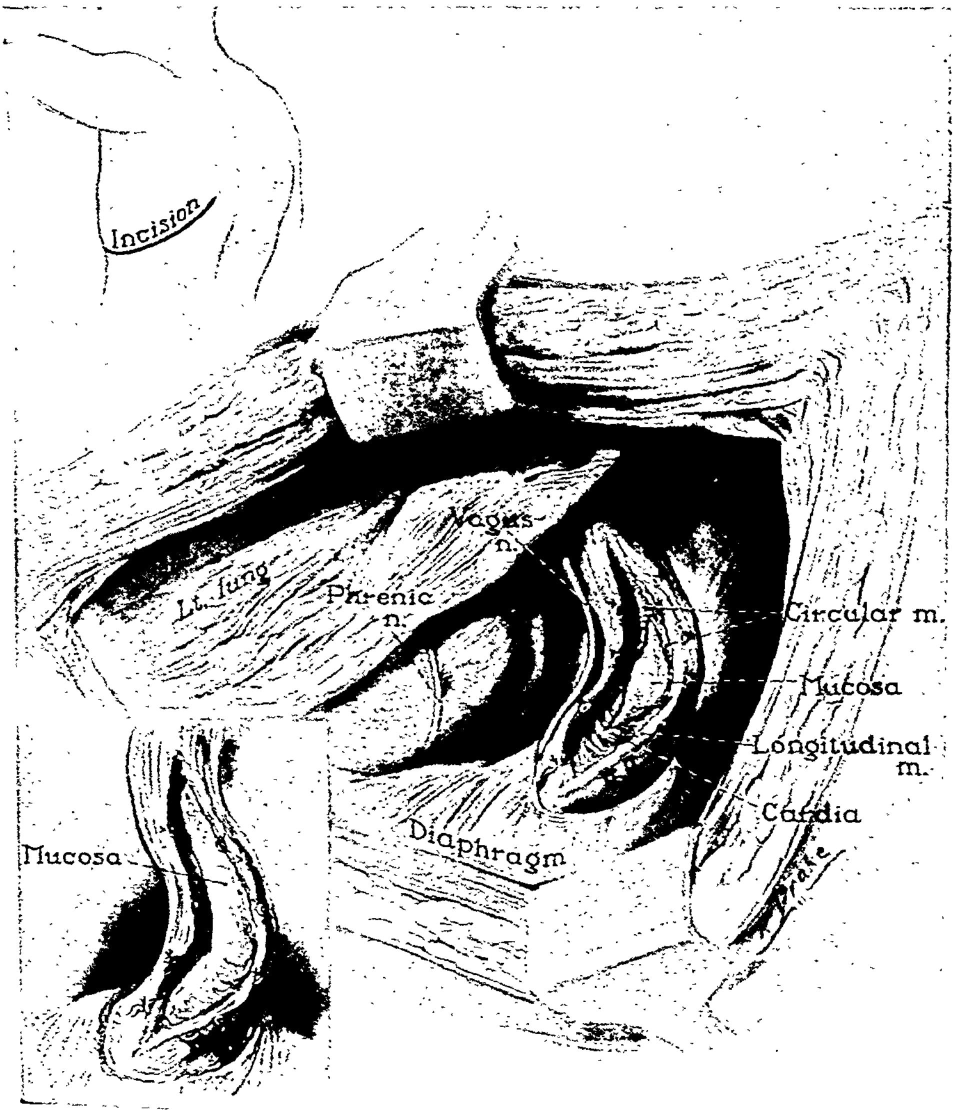

Fig. 9.—Technique of extramucous esophagocardiomyotomy (Zaaijer modification of the Heller procedure), instituted in Cases 3 and 4. Transthoracic approach.

walls of the stomach to the mucous membrane. The circular layers of muscle of the esophagus and cardia are usually very thin and often somewhat adherent to the underlying mucous membrane at the cardia. This circular layer must be separated with great care to avoid perforation of the mucosa. The blood vessels are larger and more numerous in the cardia and stomach than in the esophagus. Many of these vessels require a transfixing suture. The length of the incision depends on the amount of narrowing and constriction of cardia and lower part of the esophagus but is at least 10 cm. The mucous membrane is permitted to protrude throughout the entire extent of the longitudinal incision through the constricted area of esophagus and cardia. The cardiac end of the stomach is replaced into the abdomen. The incision in the esophageal ring is sutured with interrupted silk and the incision in the mediastinal pleura over the lower part of the esophagus is closed with interrupted catgut. The chest wall is closed and the pleural cavity is drained with an air-tight catheter and controlled suction for two to three days.

COMMENT ON SURGICAL RESULTS

From a review of our twenty-three surgical cases it is impossible to draw any definite conclusions concerning the best operative procedure. Twenty-three cases is a relatively small group and eight different operative procedures were utilized. The different procedures were used in from one to five cases each. These numbers are not only too small but the cases are not sufficiently uniform to compare the results of the different operations. However, certain deductions are justifiable: (1) The best results were obtained in those cases in which there were *only moderate secondary changes of dilatation and thickening* of the wall of the esophagus. (2) The poorest results were obtained when there was marked dilatation especially when it was associated with retention and elongation of the esophagus regardless of the type of operative procedure.

Comparison of the results revealed that transgastric retrograde dilatation or sympathectomy or both, almost always gave unsatisfactory results (poor in five cases and good in one). These procedures have not been utilized in recent years. The other five types of operative procedures utilized in the remaining eighteen cases represent the operations for cardiospasm most frequently used at this time. The results obtained from these five different operations in this series of eighteen patients were varied but essentially the same with each type of operation with the probable exception of the esophagocardiomyotomy. Some of the patients who have undergone esophagocardiomyotomy have not been operated on a sufficient length of time to permit a final evaluation of the results.

All of these operative procedures are directed toward relieving the obstruction. The immediate and early result in practically all cases regardless of type of operative procedure was satisfactory and the patient was greatly relieved because of the removal of the obstruction. In 6 of these cases, however, the long term result was unsatisfactory because of esophagitis from the free regurgitation of gastric secretions into the esophagus. In the three cases in which the result has been classified as fair, the esophagitis was of a lesser degree. The effect of gastric secretions in the esophagus has been discussed repeatedly. Maingot,[41] Barrett and Franklin,[43] and others[27, 46] have called attention to the fact that the late unfavorable results after operations for cardiospasm are due to the development of peptic ulceration in the esophagus. Late fatal hemorrhages occurred in two patients, four years and one and one-half years after operation.

The development of dysphagia in the other patients who had recurrent trouble is presumably the result of inflammatory and cicatricial changes produced by the regurgitation of gastric secretions. The mechanism is not unlike that described by Allison,[47] Olsen and Harrington[48] and others, in which incompetence of the cardiac sphincter leads to peptic esophagitis, short esophagus and esophageal stricture.

The ideal surgical operation is one in which the cardiac sphincteric control of gastric secretions will be maintained and the obstruction of the esophagus relieved. Our experience at this time with the esophagocardiomyotomy (Heller type of procedure) is not sufficient in number of cases or length of time since the operations to permit any final conclusion. The results so far, however, have been encouraging. We are hopeful that this procedure will accomplish the objective of operative treatment for cardiospasm which is the relief of the obstruction without superimposing a complication such as esophagitis that may be as serious as or more dangerous than the original condition.

COMMENT AND CONCLUSIONS

The cause of cardiospasm is not known and hence a truly rational approach to the treatment of this disease has not been evolved. Whether there is actual spasm at the physiologic sphincter of the lower part of the esophagus or whether the cardia merely fails to open, is debatable. Whatever the etiology of this disease may be, the present methods of treatment are directed toward the relief of obstruction at the cardia.

Our study and experience with 601 cases have led us to formulate certain opinions and conclusions. These are as follows:

1. Exact diagnosis is important. Cardiospasm must be differentiated from diffuse spasm of the esophagus. Diffuse spasm does not respond favorably to either hydrostatic dilatation or surgical treatment.
2. Hydrostatic dilatation at present is the treatment of choice for most patients with cardiospasm. Relief of dysphagia for periods of four to sixteen years was obtained from a single course of dilatation in 60.1 per cent of our 452 traced cases. If we are given the opportunity of treating those patients a second time for whom an initial course of treatment by hydrostatic dilatation did not permanently relieve dysphagia, the percentage of satisfactory results can be increased to 75 per cent. However, if satisfactory results are not obtained with two courses of hydrostatic dilatation, additional treatment is likely to be of little avail. We must anticipate that about 20 per cent of the patients cannot be permanently relieved by dilatation.
3. Successful dilatation in the case of properly diagnosed cardiospasm depends on (a) accurate placement of the dilator in the cardia, and (b) severe forceful dilatation of the physiologic sphincter by an expanding dilator. A previously swallowed thread permits accurate placement of the dilator and adds to the safety of the procedure.

4. Surgical treatment should be considered for those patients who fail to respond to two or three courses of dilatation therapy. Every effort should be made to carry out surgical treatment before marked changes in the esophageal wall have taken place. Less favorable results can be expected when surgical therapy of the advanced cases is undertaken.

5. Surgical correction of cardiospasm may destroy the sphincteric property of the cardia. The regurgitation of gastric secretions may result in peptic esophagitis and account for poor surgical results.

6. The Heller operation appears to be the most advantageous surgical procedure. This procedure permits the cardia to retain some of its sphincteric property.

7. If the Heller operation is not feasible and one of the operations is performed which destroys the sphincteric action of the cardia, then an effort must be made to reduce the acid-production of the stomach.

8. The results of the surgical management of cardiospasm at the clinic have not been very favorable for two reasons: Most of our patients selected for surgical treatment had far advanced cardiospasm for which dilatation therapy was impossible or had failed to give relief. Most of the operative procedures carried out permitted reflux of gastric secretions into the esophagus. The passage of time has given us an opportunity to determine the late results in most of our cases. Peptic esophagitis and ulceration were responsible for most of the poor results.

9. Treatment of cardiospasm, either by hydrostatic dilatation or by surgical means, can be carried out with little risk.

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VARIATIONS IN THE BRONCHOVASCULAR PATTERNS OF THE RIGHT LOWER LOBE OF FIFTY LUNGS

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IN A previous article by Smith and Boyden (1949) the principal variations in the distribution and arrangement of the segmental bronchi were established, first by injections and then by dissection of injected specimens. At that time, even if the technique had permitted, it would not have been feasible to analyze the vascular patterns owing to their complexity and the necessity of first mastering the bronchial variations. Nevertheless, it was recognized that a knowledge of the arrangement of arteries and veins is as essential to segmental resection or lobectomy as familiarity with bronchial patterns. Indeed, the present study has shown that such malarrangements as the splitting of the medial basal bronchus by the basal branches of the inferior pulmonary vein may prevent the resection of that segment even though the bronchogram might not suggest the danger of resection to the uninitiated observer.

To obtain the necessary information, fifty right lower lobes were removed from cadavers and dissected completely. Accurate drawings were made of the paravertebral view of the medial basal segment (B^7), of the dissected anterior surface of the lobe, of the lateral view—for optimum exposure of the arteries—and of the opened bronchial tree.

Obviously, it was necessary again to identify the bronchial variations. New information has been gained because dissection of soft cadaver material is more accurate than dissection of specimens filled with gelatin, and in addition percentages could be calculated on the basis of 100 specimens, those of Smith and Boyden's being added to our own.

THE GENERAL VENOUS PATTERN

Since the veins lie between adjacent bronchi and offer cleavage planes by which segments and subsegments can be separated from one another, it is advantageous to start with the venous pattern. With minor exceptions the veins of the right lower lobe drain into the inferior pulmonary vein. As shown in Plate 1—a diagram of the dissected anterior surface of a truncated right lower lobe—the inferior pulmonary has two principal tributaries, a *superior vein* (V^6) draining the superior segment, and a *common basal* vein draining the basal segments.* The latter is formed by the union of a *superior basal* vein, draining primarily segments B^8 and B^9, and an *inferior basal vein* draining

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*These correspond to the truncus dorsalis and truncus ventralis of Adachi (1933), and to the ramus superior and ramus inferior of Herrnheiser and Kubat (1936). Our terms are designed to emphasize the relation of these veins to the superior and the basal segments.

primarily segment B^{10} (or segments B^{9+10}). This pattern occurs in 78 per cent of specimens. The anomalous patterns found in the remaining 22 per cent will be discussed at the end of this section (Table III).

The varying composition of the basal veins is shown in Table I.

TABLE I. PATTERN OF BASAL VEINS

SUP. BASAL	INF. BASAL	PERCENTAGE	
1. Uncomplicated types			38%
V^{8+9}	V^{10}	32%	
V^{8}	V^{9+10}	6%	
2. "Split" types			62%
V^{8+9}	V^{9+10}	44%	
$V^{8,9,10}$	V^{10}	14%	
V^{8}	$V^{8,9,10}$	4%	

In the uncomplicated types all of V^8, or V^9 or V^{10} is found to be a tributary of either the superior or the inferior basal vein. The most common pattern (32 per cent) is that shown in Plate 1. In the "split" type, part of either V^8, or V^9 or V^{10} joins both the superior and inferior basal veins. Here, the commonest arrangement (44 per cent) and, therefore, the prevailing pattern for the lobe, is that in which there are two V^9s, one joining the superior basal, the other the inferior basal vein. Unfortunately, none of the split types are shown in the illustrations, but the tabular presentation indicates the futility of giving descriptive names to the larger tributaries (cf. Melnikoff, 1922, and Herrnheiser and Kubat, 1936).

At this point, it is desirable to point out that the main trunks of the basal veins lie deep to the anterior surface of the lobe and that the key to their dissection are the intersegmental branches of B^8b (Fig. A, Plate 2). These superficial veins separate the medial basal (B^7) from the anterior basal segment (B^8). If one dissects obliquely medialward along the plane of these veins to V^9 and V^{10}, the medial basal segment can be lifted medialward thus exposing the deeper veins and bronchi. If one were to apply to the median basal the principles of segmental resection recently developed by Overholt, Woods, and Ramsay, the superficially placed A^7 (medial basal artery) would be ligated first (Fig. A, Plate 2), then B^7 (the medial basal bronchus). As the cut ends are pulled forward, the small segmental veins of V^7 would be ligated and then the plane of separation from the deeper segments developed along the anterior surfaces of V^8b, V^9 and V^{10} (Fig. E, Plate 2).

The veins of the medial basal segment (V^7) have not been included in the above classification for the reason that they are small veins ranging from one to six in number. If there is only one, it usually empties into the inferior basal vein or its junction with the superior basal (Plate 1) or directly into the inferior pulmonary vein (Plate 2). When V^7 is multiple, the orifices of V^7a may be scattered along the superior basal vein, and those of V^7b along the inferior basal, or located upon either of the large veins (as in Plate 6, #41, A).

Similarly the veins from the subsuperior zones (V^* and VX^*) have not been included in the above table since they are variable and may empty into either the proximal or more distal portions of the great veins. Using V^* as an example, it terminates in the twenty-eight specimens in which it occurred as follows: once on the inferior pulmonary, coordinate with V^6 (cf. Plates 2 and 3); twice on V^6c or V^6b; six times on the common basal; three times on the proximal portion of the superior basal; five times on the proximal portion of the inferior basal; seven times on the distal portion of the superior basal or its branches; and four times on the distal portion of the inferior basal or its branches. In sum, it empties into the proximal portions seventeen times, and into the distal portions eleven times. (Incidentally, the termination of V^* in the common basal was noted seven times by Adachi. He named this vein the distal truncus dorsalis in contrast to V^6 which he called the proximal truncus dorsalis.)

As to the course of these veins from the posterior sector of the lobe to the hilar region, one may say that almost always those which terminate in the proximal portions of the great veins pass medial to the posterior basal bronchus, B^{10} (cf. V,* lateral views, Plate 3, and #24, Plate 5). Those which terminate in the distal portions pass between B^8 and B^9 or between B^9 and B^{10} (cf. VX,* Plate 6, #41).

THE RELATION OF THE BASAL VEINS TO THE MEDIAL BASAL SEGMENT

Probably the basal segment which is least understood, clinically, is the medial basal (B^7). Doubtless this is due to the smaller size and the somewhat concealed position of its bronchus. In anteroposterior bronchograms it is hard to see. Brock (1945) has pointed out its arched appearance in lateral bronchograms, but few, if any, observers have paid attention to its branches, the anterior and medial rami (B^7a and B^7b). Yet, as a result of this scrutiny of fifty specimens, we have come to believe that it is the key to an understanding of the basal half of the lobe.

In analyzing the variations of this segment we have utilized a landmark which Dévé named "the hornlike projection of the cardiac segment." This is the anteroparavertebral ridge which separates the anterior from the paravertebral surfaces of the lobe. In preserved specimens, it occupies a position slightly medial to the medial boundary of the fossa of the inferior vena cava ("Ridge," Plate 4, Type II). Occasionally it coincides with that boundary. The relation of the pulmonary ligament to this ridge is variable. It may arise from the ridge or lie beyond it on the paravertebral surface. (In the latter case, one may speak of a "retro-pericardiac recess" between the ridge and the ligament.) Because the position of the ligament is variable it may not be used to define the anterior limit of the paravertebral surface. Posteriorly (Plate 7), that surface is limited by the costoparavertebral margin (Smith and Boyden).

Four types of the medial basal bronchus may be recognized (see diagrams, Plate 4): Type I, that in which B^7 is distributed to both anterior and paravertebral surfaces and passes superficial to the basal veins. This is the prevailing pattern (34 per cent); Type II, that in which the distribution is restricted to the anterior surface (22 per cent).* This represents only a slight variation

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*In all four types deeper branches of the rami pass to the corresponding diaphragmatic surface, but for the purpose in hand, no further mention of these is necessary.

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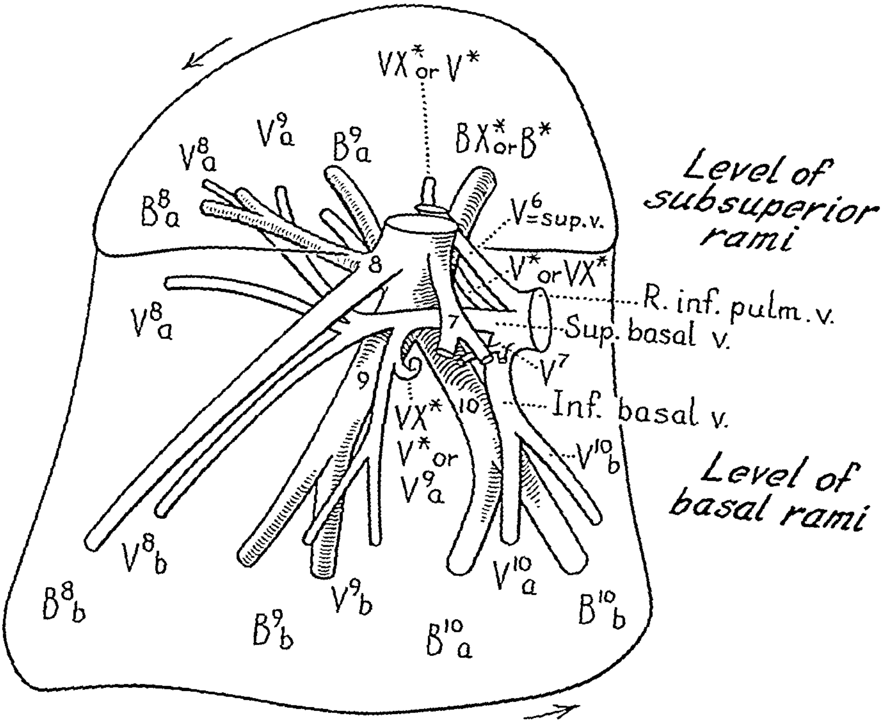
EXPLANATION OF PLATES

(Sketches, by E. A. Boyden, rendered for publication by Lawrence B. Benson)

Plates 1-7.—A series of drawings illustrating variations found in the vascular patterns of the right lower lobe of fifty dissected lungs.



Scheme showing common arrangement of veins in basal segments

Plate 1.—Diagram of truncated right lower lobe to show venous pattern and method of numbering branches of basal veins. The vein on the counterclockwise side of a given bronchus (cf. direction of arrows) receives the number of that bronchus, viz., V^8b is the vein on the counterclockwise side of B^8b. In the left lower lobe corresponding veins would lie on the clockwise side (Boyden, 1945). B^7, Medial basal bronchus; B^8a, B^8b, lateral and basal rami of anterior basal bronchus; B^9a, B^9b, lateral and basal rami of lateral basal bronchus; $B^{10}a$, $B^{10}b$, laterobasal and mediobasal rami of posterior basal bronchus; B^*, BX^*, subsuperior and accessory subsuperior bronchi.

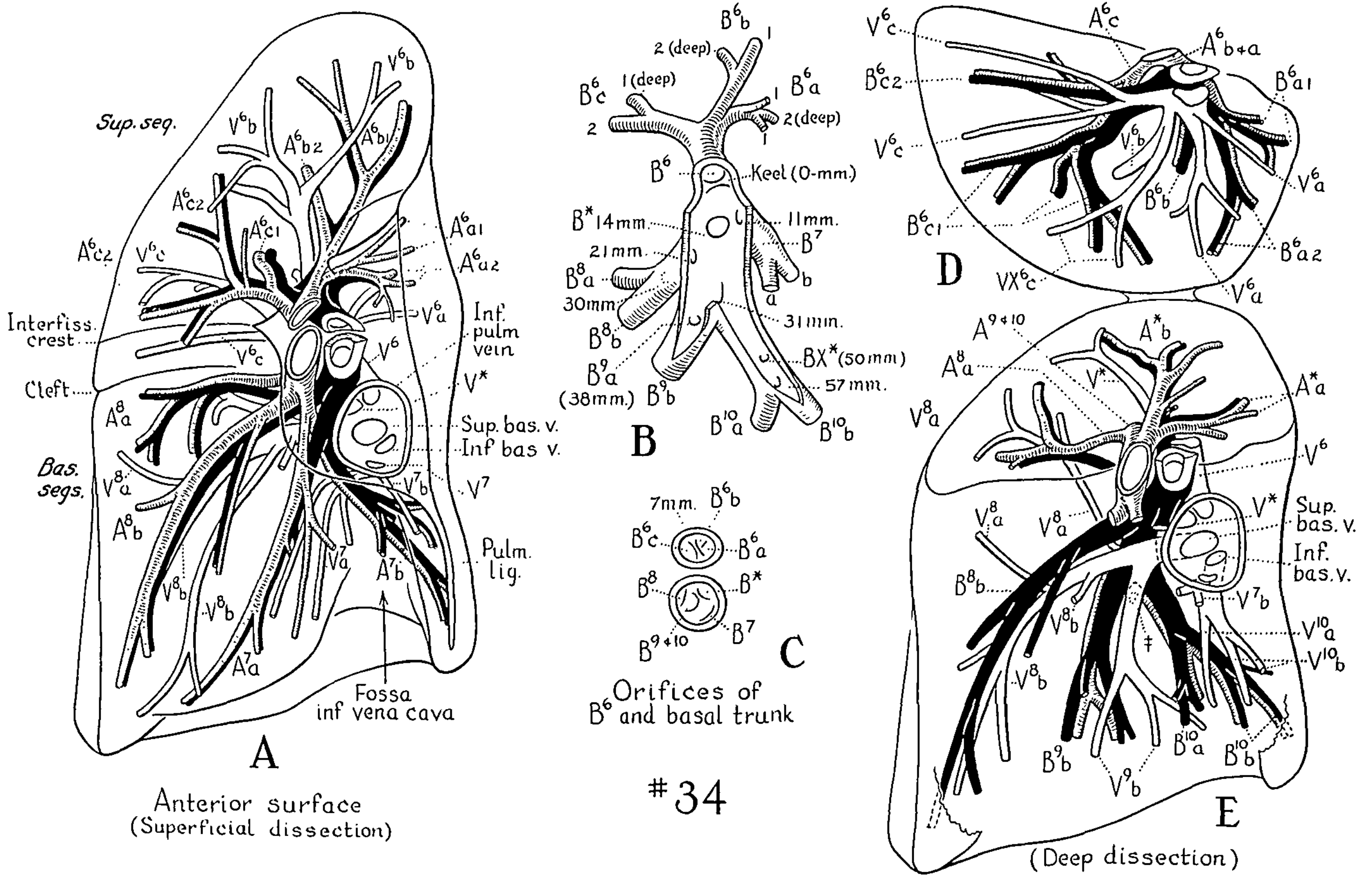
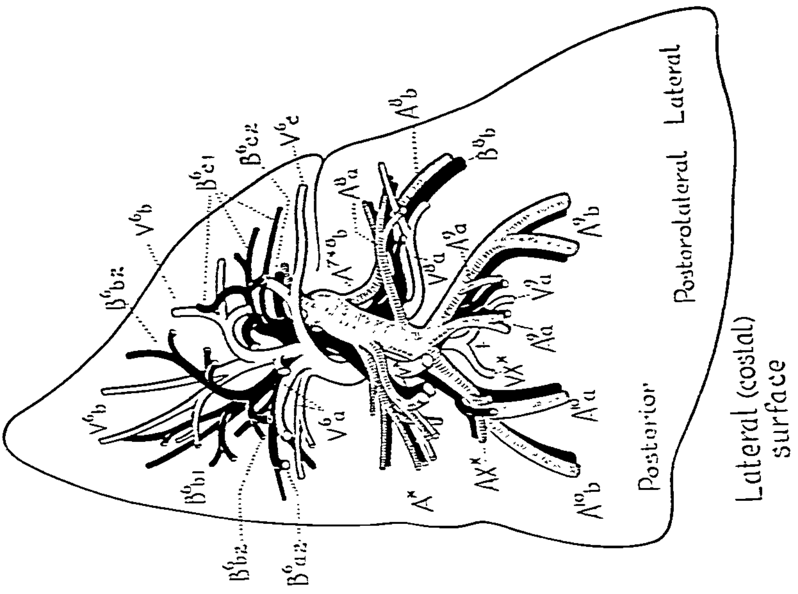


Plate 2.—Dissection of typical specimen (#34) classified as Type I (see Plate 4). Fig. *B*, opened bronchial tree; *C*, inverted bronchoscopic views; *D*, superior segment tilted back to show intersegmental veins. *Black* indicates bronchi; *red*, the pulmonary arteries; *blue*, the pulmonary veins. For names of segmental bronchi, see Text Figures 1 and 2 (cf. Boyden, 1949).



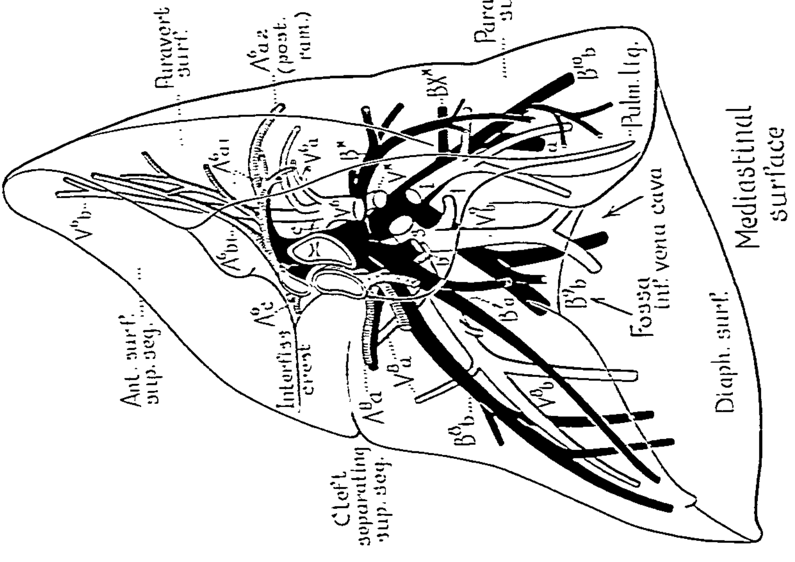
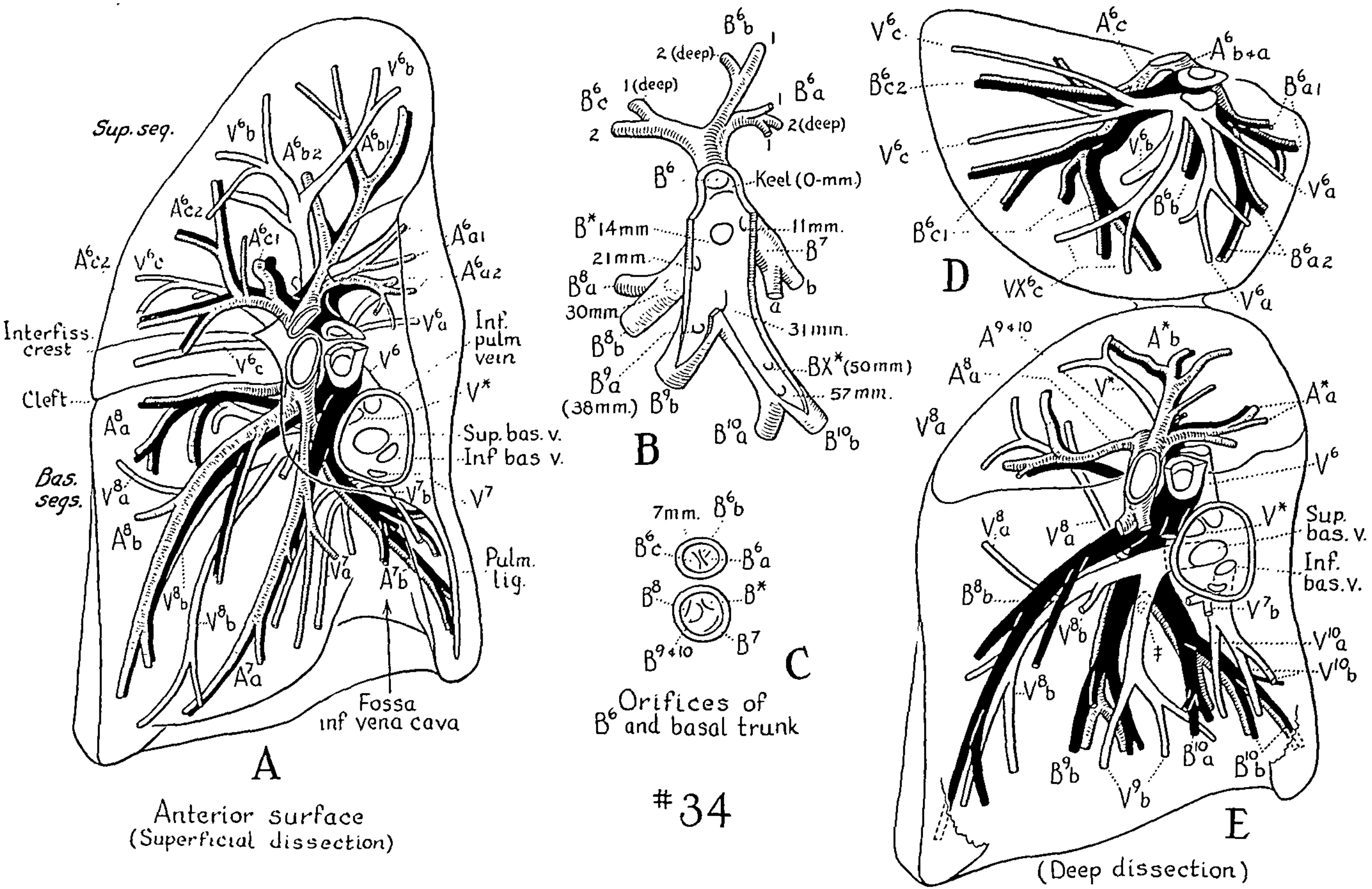


Plate 3.—Mediastinal and lateral views of the dissected specimen shown in Plate 2.



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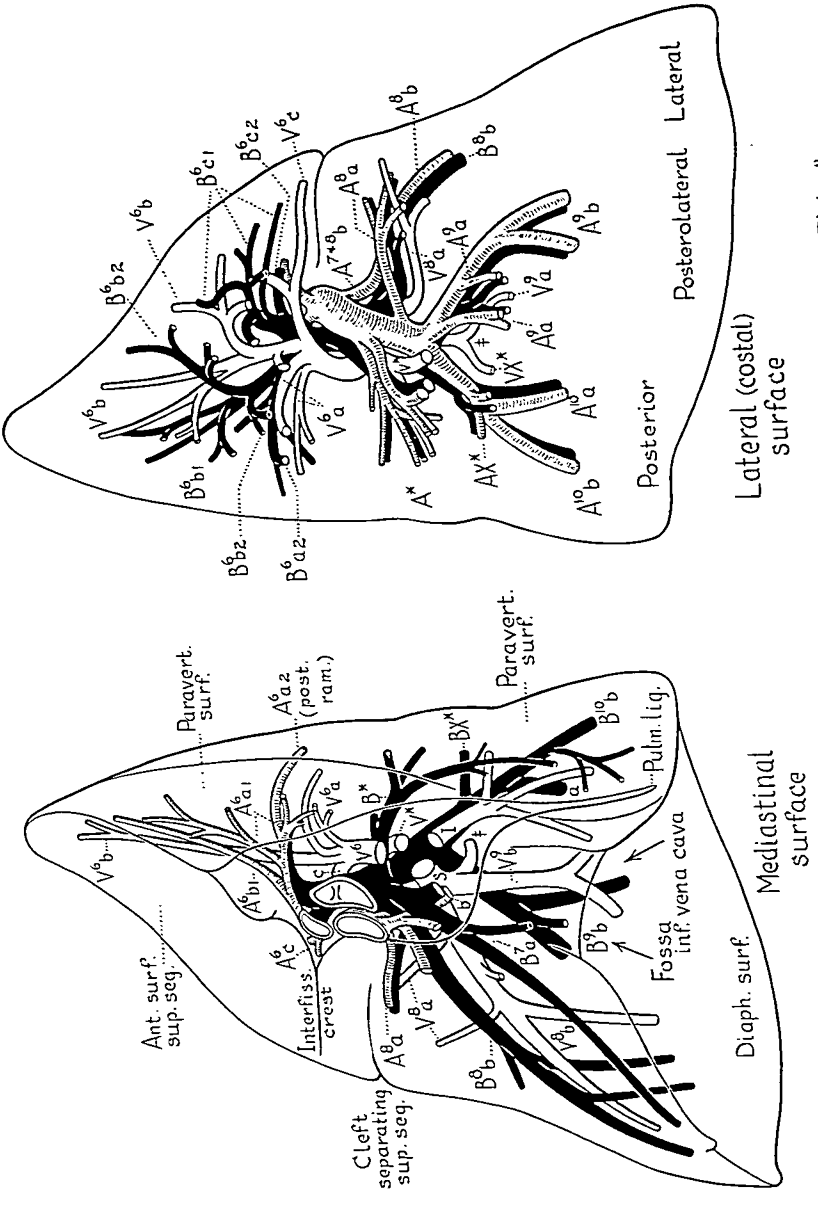
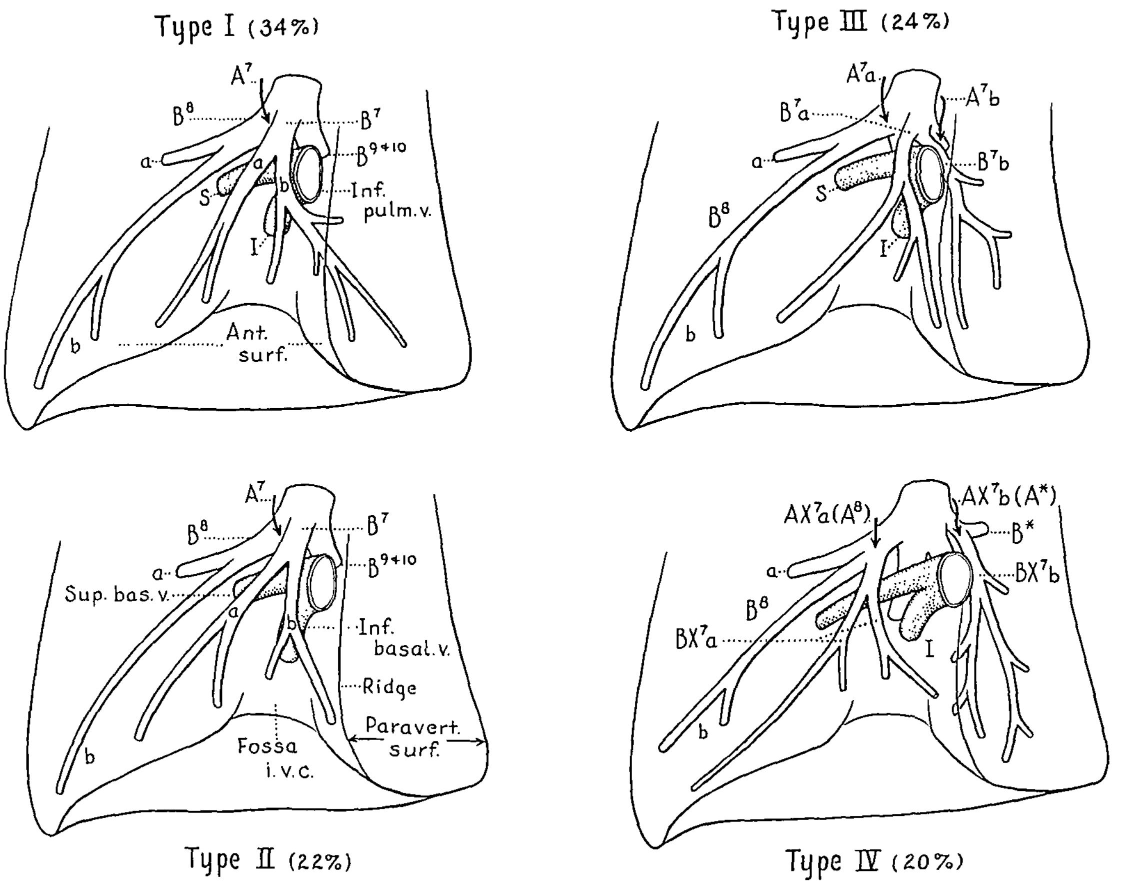


Plate 3.—Mediastinal and lateral views of the dissected specimen shown in Plate 2.



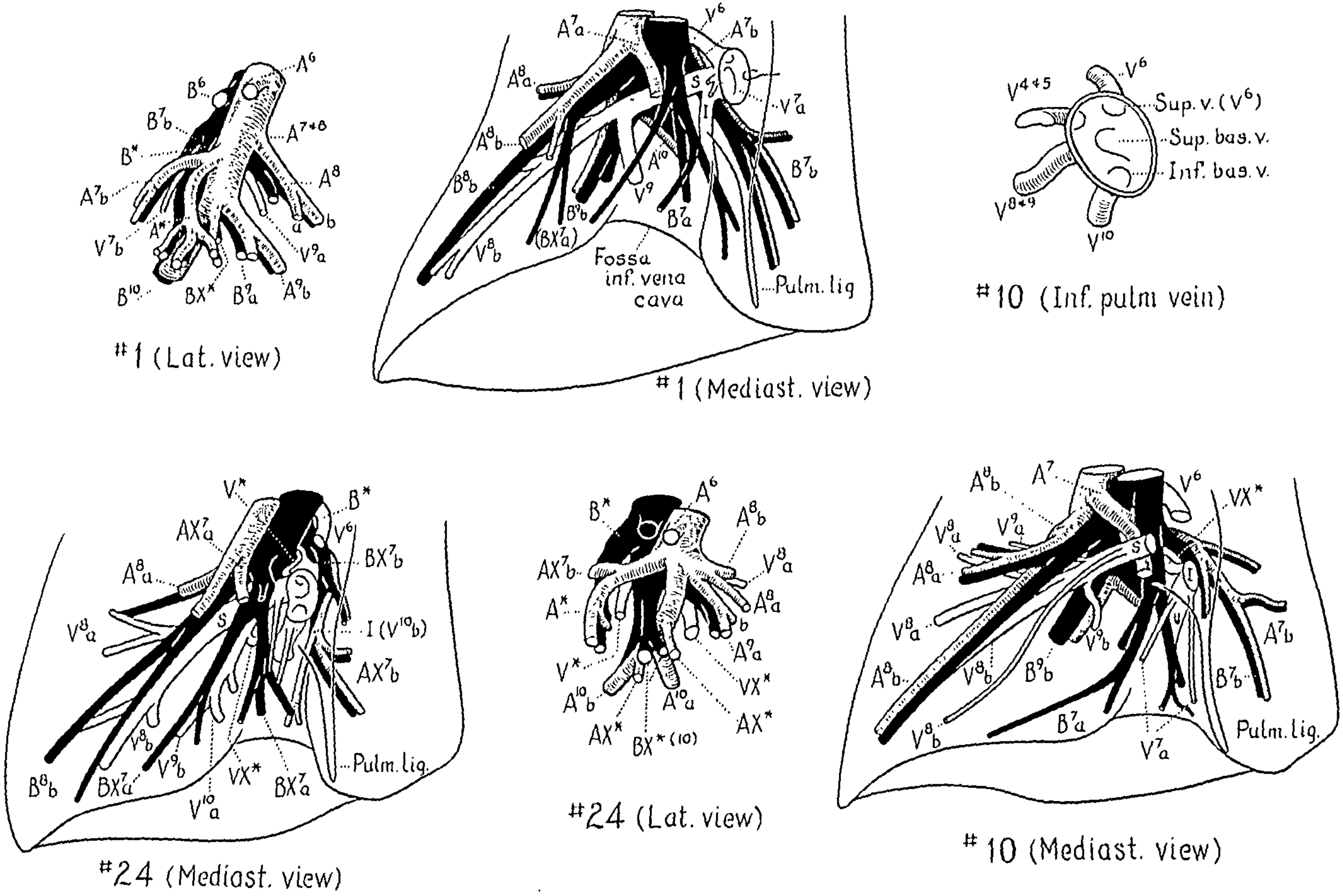
Type I (34%)

Type III (24%)

Type II (22%)

Type IV (20%)

Plate 4.—Diagrams illustrating four types of arrangements of the branches of the medial basal bronchus, based on the origin and distribution of rami and their relation to the basal veins. Arrows indicate position of arteries. *B^7a*, anterior ramus; *B^7b*, medial ramus; *BX^7a*, *BX^7b* displaced anterior and medial rami.



#1 (Lat. view)

#1 (Mediast. view)

#10 (Inf. pulm vein)

#24 (Mediast. view)

#24 (Lat. view)

#10 (Mediast. view)

Plate 5.—Sketches of three dissected specimens displaying arrangement of structures in specimens illustrating Type III (#1), a modified Type III (#10) and Type IV (#24). The vein drawn in upper right corner (#10) is not characteristic of Type III, but illustrates an unusual anomaly.

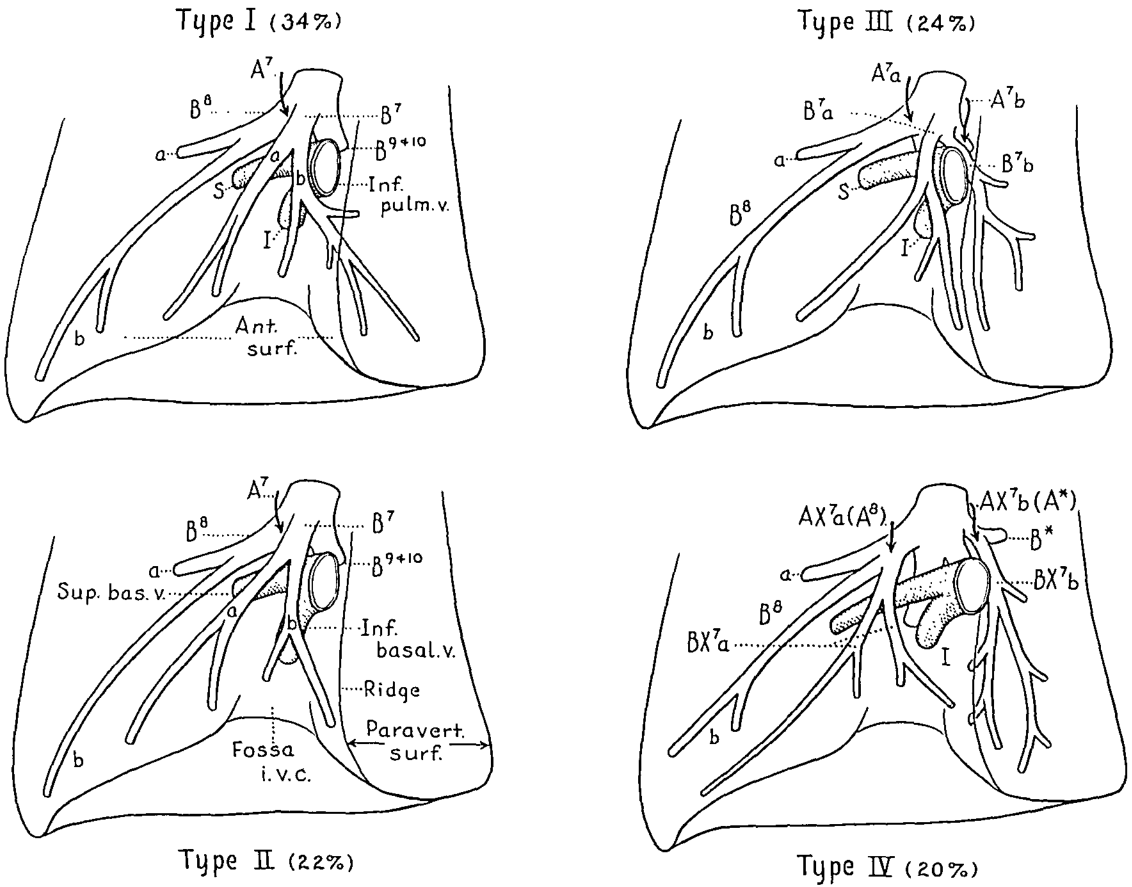
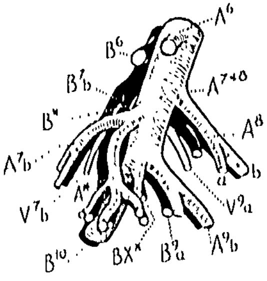
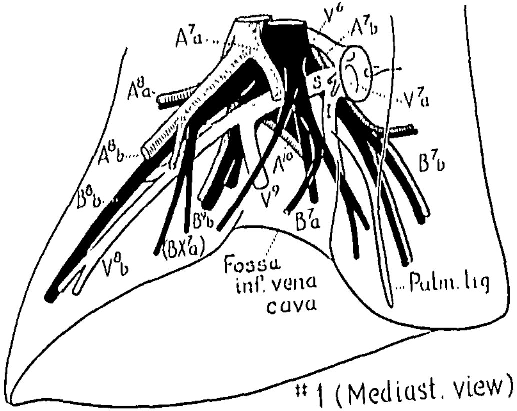


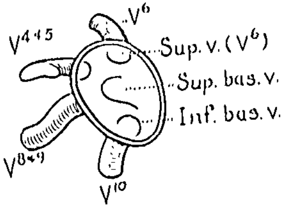
Plate 4.—Diagrams illustrating four types of arrangements of the branches of the medial basal bronchus, based on the origin and distribution of rami and their relation to the basal veins. Arrows indicate position of arteries. *B^7a*, anterior ramus; *B^7b*, medial ramus; *BX^7a*, *BX^7b* displaced anterior and medial rami.



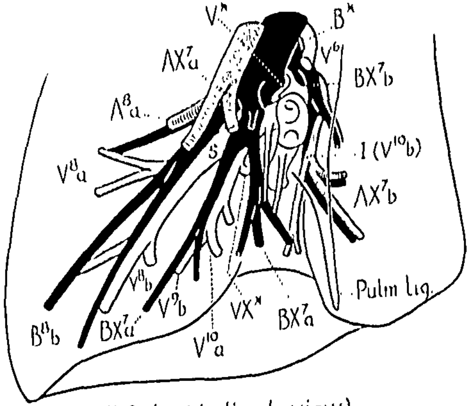
#1 (Lat. view)



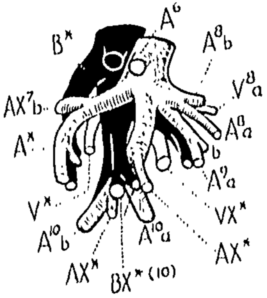
#1 (Mediast. view)



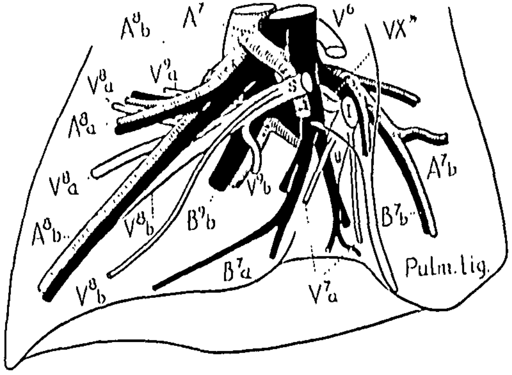
#10 (Inf. pulm vein)



#24 (Mediast. view)

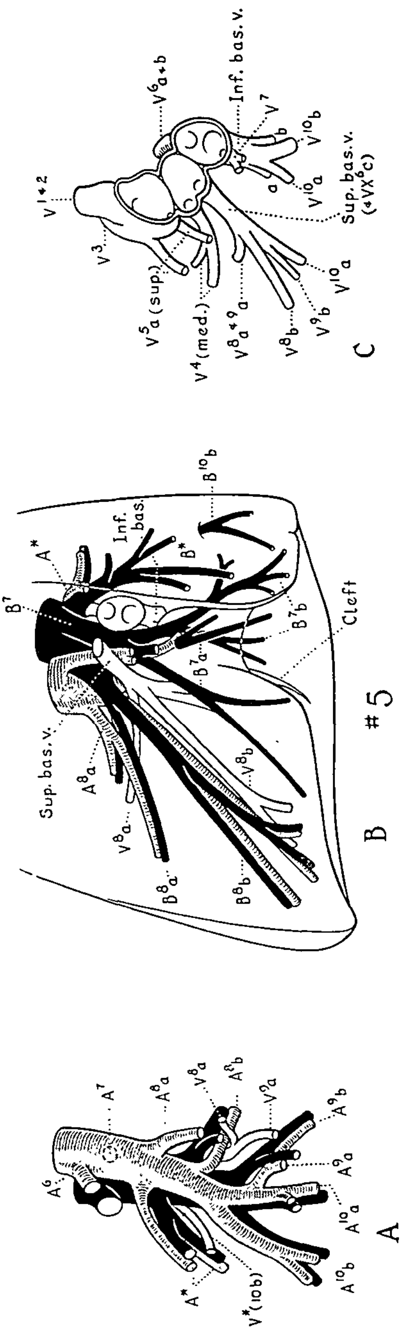


#24 (Lat. view)

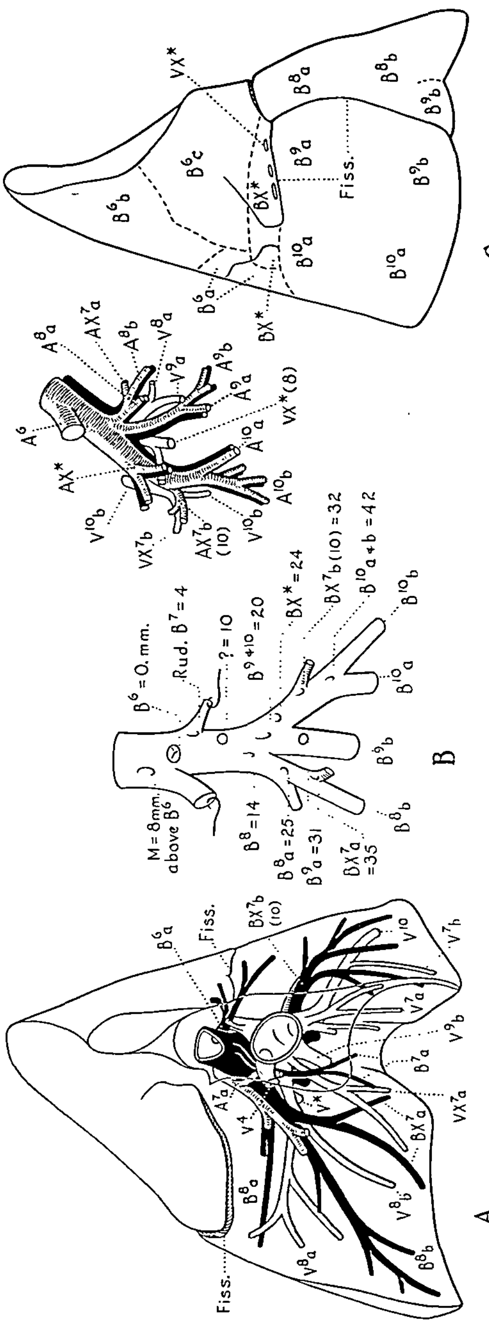


#10 (Mediast. view)

Plate 5.—Sketches of three dissected specimens displaying arrangement of structures in specimens illustrating Type III (#1), a modified Type III (#10) and Type IV (#24). The vein drawn in upper right corner (#10) is not characteristic of Type III, but illustrates an unusual anomaly.



#5



#41

Plate 6.—Dissected specimens illustrating arrangement of structures in a modified Type I (#5) and in a type that is transitional between I and IV (#41). In both cases the inferior pulmonary vein is atypical. Fig. 41 C demonstrates that fissures do not necessarily separate segments but may develop between rami of a segment (e.g., B^9b). V^*, in Fig. A, should read VX^*.

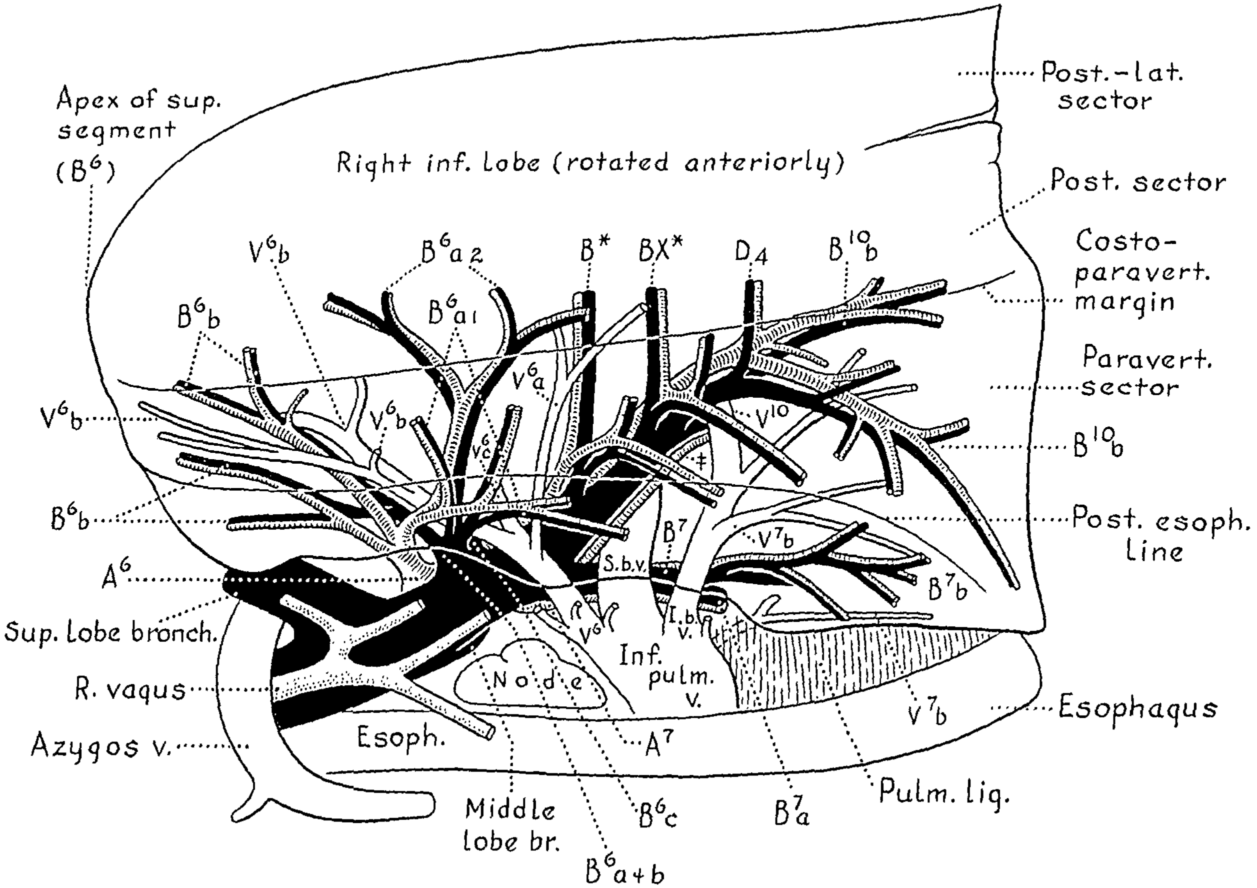


Plate 7.—Posterior surface of right lower lobe, dissected in situ after the lobe has been elevated (Specimen #14154). Note series of dorsal rami—B^*, B^*, BX^* and D_4 and their *paravertebral branches*. *S. b. v.*, superior basal vein; *I. b. v.*, inferior basal vein; ‡artery to $B^{10}a$ arising atypically from A^7.

from the normal pattern; Type III, that in which the bronchus arises from a more medial position, and bifurcates almost immediately around the basal veins (24 per cent). In this type the B^7a branch is superficial to the veins and is distributed anteriorly, the B^7b branch is deep to the veins and is distributed paravertebrally; Type IV, that in which B^7 is absent as such (20 per cent) and the branches which represent it arise independently from adjacent bronchi—BX^7a, from the anterior basal (B^8 or B^8b), and BX^7b from the subsuperior (B^* or BX^*) or, rarely from the posterior basal (B^{10}), as at #41 A, Plate 6.[†]

Exact drawings of specimens illustrating these types are shown in the following plates: Type I at A, Plate 2; a modified Type I at B, upper row of figures, Plate 6 (here the superior basal vein passes superficial to B^7, but the rest of the inferior pulmonary is normally situated, i.e., medial to the stem of B^7); Type III, upper row of Plate 5; a modified Type III, in lower right corner of Plate 5 (here, the superior basal vein passes superficial to B^7a, but the inferior basal passes between B^7a and B^7b); Type IV in the lower left corner of Plate 5. Finally, specimen *#41* in Plate 6 is of special interest as a transitional type. B^7 is represented by a rudimentary stem (B^7a). If this be ignored as negligible, then the specimen falls into Type IV with a BX^7a ramus arising from B^8b and a BX^7b ramus originating on B^{10}.

Before discussing the resectability of these types it is desirable to summarize the distribution of B^7. First, the line of division between B^7a and B^7b shifts in the different types (Plate 4). Prevailingly (Type I), it lies in the fossa of the inferior vena cava. In Type II, the line moves toward the lateral lip of the fossa, and in Types III and IV to the medial lip—specifically the anteroparavertebral ridge.

Second, the lateral limit of B^7, in Types I and II, was found to lie more than half way along the anterior margin from ridge to lateral margin of the lobe, in 42 per cent of specimens.

Third, B^7 was found to be absent as such in 20 per cent of specimens, to have a wholly anterior distribution in 22 per cent, and to supply both anterior and paravertebral surfaces in 58 per cent. Smith and Boyden's figures for fifty specimens were, respectively, 14 per cent, 8 per cent, and 78 per cent, but since in that article the anterior distance was measured from the variable pulmonary ligament, only the percentages for Type IV can be combined; that is, one can say that in 100 specimens B^7 was absent as such in 17 per cent.

From the foregoing study, it would appear that resection of the medial basal segment—either by itself, for localized bronchiectasis (Scannell), or in conjunction with middle lobectomy (Brock, 1950)—is technically feasible in Types I and II, but not in Type III. For in that type the basal veins lie between the rami of B^7 and ligation of them would seem to be fraught with hazard. Furthermore, in three-fourths of these specimens (as #1, Plate 5) the artery of B^7b originates posteriorly and can be reached only with difficulty.

The question thus arises as to how Type III may be identified preoperatively in bronchograms. Three criteria are submitted. First, B^7 originates more medi-

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[†] The letter "X" is added to a ramus to indicate that it is displaced in origin and therefore accessory to the bronchus from which it does arise (Boyden, 1945).

ally than usual on the lower lobe bronchus. Second, it bifurcates almost at once into its principal rami.* Third, the fork is wider than usual in order to permit passage of the basal veins. Once these points have been established, segmental resection of the deeper ramus *(B^7b)* would seem to be inadvisable.

THE VEINS OF THE SUPERIOR SEGMENT *(V^6)*

The primary drainage of this segment is effected by the superior vein *(V^6)*. In 86 per cent of specimens it swings around the medial side of the lower lobe bronchus, between the superior and subsuperior bronchus *(B^7* and B^*, Plates 3 and 7), to enter the inferior pulmonary vein on the medial side of B^7 (Fig. *A*, Plate 2). In another 12 per cent it enters the inferior pulmonary at a lower level, either passing between B^7a and B^7b (6 per cent) or beneath B^* (6 per cent), as in Plate 6 *(#5)*. Finally, in 4 per cent (2 per cent being included in the preceding category) the segment is drained by two separate veins *($V^6a + b$* and *V^6c;* see items indicated by a dagger, in Table III).

In addition to this primary drainage there is frequently a secondary channel *(VX^6)* which drains into the posterior upper lobe vein *(V^s)*. This arises from the paravertebral or superior portion of the segment—an area that is frequently fused to the upper lobe. Boyden and Scannell found such a vein in 54 per cent of specimens.

The tributaries of V^6 lie between the three rami of the superior segment and adjacent segments. Before discussing these it is necessary to review the bronchial patterns in the superior segment (see Fig. B, Plate 2). The variations are summarized in Table II, our figures having been combined with those of Smith and Boyden to give statistics based on 100 specimens.

TABLE II. SUMMARY OF THE SUPERIOR SEGMENTAL BRONCHUS *(B^6)*

	SMITH AND BOYDEN (50 SPEC.)	FERRY AND BOYDEN (50 SPEC.)	TOTAL (100 SPEC.)	
I. B^6 arises as a single stem				
1. Bifurcates into	90%	88%		89%
B^6a+b and B^6c	86%	72%	79%	
B^6b+c and B^6a	--	8%	4%	
B^6a+c and B^6b	--	2%	1%	
bizarre patterns	4%	6%	5%	
2. Trifurcates into B^6a; B^6b; B^6c	6%	4%		5%
II. B^6 arises as two separate stems				6%
1. B^6a+b; B^6c	2%	6%	4%	
2. Miscellaneous patterns	2%	2%	2%	

A survey of Table II indicates that the prevailing pattern is that shown at B in Plate 2—namely, a bifurcation into $B^6a + b$ and B^6c.

Three new observations have emerged from this study. The first records additional modes of bifurcation. The second establishes the almost constant presence of the deep branch of B^6a (98 per cent). Smith and Boyden reported it in only 38 per cent of specimens, but owing to the difficulty of dissecting specimens

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*The average distance from the keel of B^7 to the keel which separates the rami is 0.6 cm. In specimens of Types I and II, the average distance is 1.35 cm.

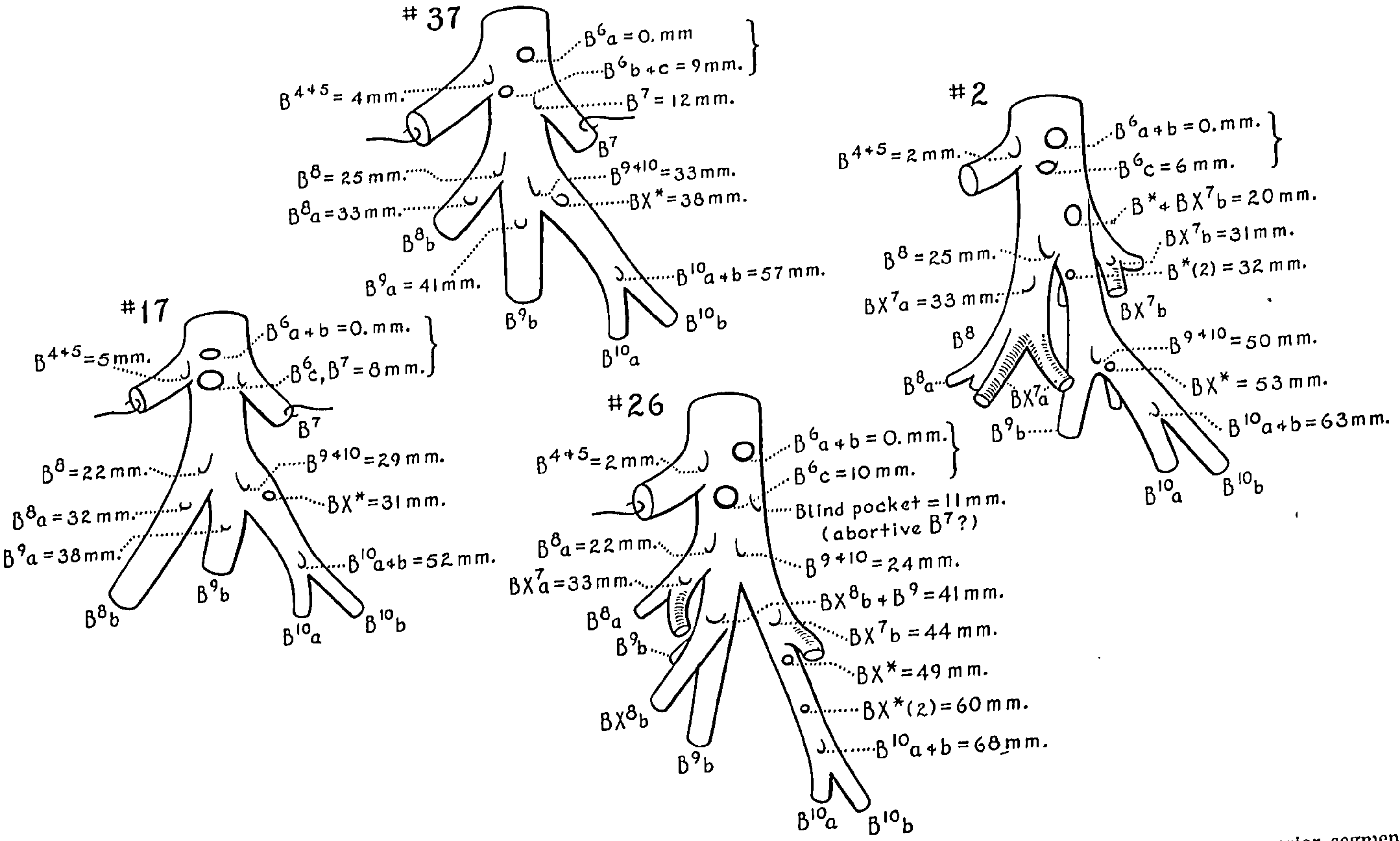

Fig. 1.—Sketches of the bronchial trees of four anomalous right lower lobes. Each has two orifices for the bronchi of the superior segment (B^6). The trees are drawn with B^7 retracted to the right and B^8 to the left. The keels of the bronchi are shown as if seen through transparent walls. Specimen #17 belongs to bronchial Type I (cf. Plate 4), #37 to Type III, and #26 and #2 to Type IV. (For arterial patterns of #17 and #26, see Fig. 2.) B^{4+5}, middle lobe bronchus; B^6a, b, c, medial, superior and lateral rami of superior segment; B^7, medial basal bronchus; BX^7a, BX^7b, displaced anterior and medial rami of B^7; B^8a, B^8b, lateral and basal rami of anterior basal bronchus; B^9a, B^9b, lateral and basal rami of lateral basal bronchus; $B^{10}a$, $B^{10}b$, laterobasal and mediobasal rami of posterior basal bronchus; B^*, subsuperior bronchus; BX^*, accessory subsuperior bronchus.

filled with gelatine it is believed that this deep stem was often missed. In any event, this branch *(B^6a2)* is the largest component of the medial ramus (cf. D, Plate 2, and "Lateral Surface," Plate 3). It swings around horizontally beneath the paravertebral surface in a plane just above the basal segments and extends to the posterior sector of the lobe and sometimes beyond that. Occasionally, it may arise separately (see bronchus indicated by a double dagger in Smith and Boyden's Plate 1).

The third observation deals with an increase in the number of cases of double origin of B^6. Four specimens were found (Fig. 1). In each case the opening of the middle lobe bronchus *(B^{4+5})* was located on a level between the two orifices of B^6. The distance between the orifices of B^6 ranged from 6 to 10 millimeters. Curiously enough Specimens #2 and #26 each had three independent arteries to the segment, while #17 and #37 had just two (Fig. 2).

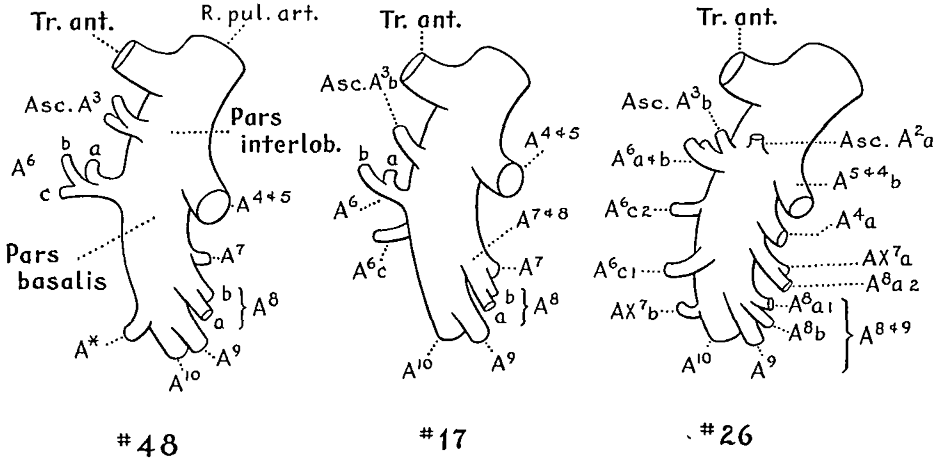

Fig. 2.—Sketches of the right pulmonary artery of three specimens. Specimen 48 is a representative of the prevailing bronchial Type I (cf. Plate 4). Its arterial pattern (except for A^6) is also of the prevailing type. #17 is normal except for two arteries to superior segment. #26 is atypical, having three arteries to the superior segment, displaced medial basal arteries *(AX^7a, AX^7b)* and two arteries to the anterior basal segment, one of which is associated with *AX^7a,* the other with A^9. *Tr. ant.,* truncus anterior to mediastinal surface of upper lobe; *Pars interlob.,* pars interlobaris, that portion of the pulmonary artery which appears in the depths of the interlobar fissure; *Asc. A^2a,* ascending artery to posterior ramus of anterior segment; *Asc. A^3,* ascending artery to interlobar side of the posterior segment of the upper lobe; *A^{4+5},* middle lobe artery; *A^6,* artery of superior segment; *Pars basalis,* basal artery to segments 7 to 10; *A^7,* artery to medial basal segment; *A^8,* artery to anterior basal segment; *A^9, A^{10},* arteries to lateral basal and posterior basal segments.

It is now possible to discuss the venous channels. It would simplify matters if one could say that the branching of V^6 is like that of the specimen shown in Plates 2 and 3, where three tributaries of approximately equal size *(V^6a, V^6b, and V^6c),* lying on the anticlockwise side of their corresponding bronchi, unite to form V^6. However, the prevailing pattern (60 per cent) seems to be that of a single large stem formed from V^6b and V^6c. In this group V^6a is represented by one or more small branches entering the main trunk of V^6. Such an arrangement is shown in Plate 7. (The three small veins at the base of the trunk are subpleural, perhaps bronchial, veins.)

In the remaining 40 per cent of specimens, the tributaries of V^6 tend to have a shrublike arrangement and are too complicated to classify. However, it is important to note that some of the larger veins tend to be located between the superior and the basal segments.

Again, if one were to follow Overholt's principles of resection, A^6 would be ligated first (Fig. A, Plate 2), then B^6. This would expose the main stem of V^6. Subsequently, the plane of separation between the superior and basal segments would be developed along the line of the intersegmental branches of V^6a, V^6b and V^6c (Fig. D, Plate 2). In a third of the specimens, however, the plane between the two is oblique instead of horizontal (Smith and Boyden). Frequently, also, it is undulating, with the subsuperior zone protruding into the superior segment (Fig. 41 C, Plate 6).* In this connection, it should be noted that transverse fissures do not necessarily demarcate the intersegmental plane. In Plate 6 such a fissure separates the subsuperior zone (BX^*) from the lateral basal segment (B^9) rather than from the superior segment (B^6). In the same figure (#41 C), a vertical fissure descending toward the diaphragm separates two branches of B^9b, although higher up it coincides with the intersegmental plane between the anterior basal (B^8a) and lateral basal segment (B^9a).

ANOMALIES OF THE INFERIOR PULMONARY VEIN

In the fifty specimens described in this article (and the 150 of the right lung dissected before that), no such gross anomaly as the drainage of the pulmonary veins into the systemic circulation has been encountered. (Brody records 68 such cases.) However, there were aberrant types of drainage into the inferior pulmonary vein in 22 per cent of the 50 specimens (Table III).

The common mode of formation of the inferior pulmonary (78 per cent) is by union of the superior vein (V^6) and the common basal vein. At the other extreme, is the pattern shown at *C* in the upper right corner of Plate 6. Here three vessels from the right lung join to form a common pulmonary vein. Healey and Gibbon found that in 3 per cent of 184 specimens the right lungs drained into a common right pulmonary vein. On the left side the figure rose to 25 per cent. The specimen in Plate 6 somewhat resembles three other cases by Healey and Gibbon (and two by Brantigan) in which a vein from each lobe of the right lung entered the left atrium; but in our specimen the middle vein drains not only a portion of the middle lobe but a portion of the superior segment and much of the lower lobe. Therefore, in intrapericardial ligation of veins, it would not be safe to assume that the three veins drain the respective lobes.

The second mode of formation of the inferior pulmonary vein is by union of three major veins (Table III). The simplest type is shown in Plate 7. The third mode is that in which four major veins are confluent.

These multiple types are due mostly to the downward displacement of veins not usually draining into the inferior pulmonary. Thus in 10 per cent the inferior pulmonary receives veins from the middle lobe (Plates 5 and 6; see also

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*Tobin and Zariguiey (1950) have also stressed this fact.

Boyden and Hamre, Plate 5, specimen #38); in another 4 per cent, it receives the posterior vein of the right upper lobe (V^3). This descends behind the root of the lung (see Boyden and Scannell, Plate 7, specimen #26).

TABLE III. MODE OF FORMATION OF RIGHT INFERIOR PULMONARY VEIN

I. Formed by union of *two* major veins			86%
1. Superior (V^6) and common basal vein		78%	
2. Miscellaneous combinations		8%	
$V^6 + VX^3$ and common basal**	2%		
$V^6 + V^*$ and common basal (Plate 2)	2%		
V^6a, b and common basal (V^6c from sup. basal)†	2%		
V^4 + sup. basal (with VX^6c) and inf. bas. + V^6a, b (Plate 6, #*5*)*†‡	2%		
II. Formed by union of *three* major veins			10%
V^6; sup. basal; inf. basal	4%		
V^6; V^4 or V^{4+5}; common basal‡	4%		
V^6; VX^3; common basal**	2%		
III. Formed by union of *four* major veins			4%
V^4; V^6; sup. basal; inf. basal (Plate 6)‡	2%		
V^{4+5}; V^6; sup. basal; inf. basal (Plate 5)‡	2%		

*These two drain into a common pulmonary vein (See #5C, Plate 6).
**Inferior pulmonary receives posterior vein (V^3) of upper lobe (4 per cent).
†Inferior pulmonary receives two veins from superior segment (4 per cent).
‡Inferior pulmonary receives veins from middle lobe (10 per cent).

THE GENERAL ARTERIAL PATTERN

If one looks into the interlobar fissure of an undissected right lung, one sees the interlobar portion of the right pulmonary artery (pars interlobaris) at the bottom of the fissure betwen the upper and middle lobes, partly covered by the posterior vein of the upper lobe (compare Boyden 1945, Fig. 3). If the middle lobe be reflected upward, the pars interlobaris may then be seen lying lateral and superficial to the corresponding stem bronchus (the bronchus intermedius of Ewart), Figs. E and A, Plate 2. From this site, the artery curves backward along the posterolateral aspect of the lower lobe bronchus. Accordingly, in a dissection the artery is best seen in a lateral view (Plate 3). In a postero-anterior film it has much the same slant, appearing as branched markings along the right border of the heart, as originally demonstrated in angiograms by Carvalho (1936).*

The *pars interlobaris* (Fig. 2), that part of the right pulmonary artery which lies between the *truncus anterior* (the principal artery to the upper lobe) and the middle lobe artery (A^{4+5}), gives rise to one or more ascending arteries of the upper lobe in approximately three-fourths of the lungs (Boyden and Scannell). In 52 per cent of lungs there are two middle lobe arteries (Boyden and Hamre). Liard found three in 4 per cent of specimens. The lower lobe artery is a continuation of the pars interlobaris. Almost immediately it gives off the artery to the superior segment (A^6) and then continues as the *pars basalis*, the trunk which supplies the basal segments (Fig. 2, #48). If there are no complications, A^6 is given off slightly below the level of the middle lobe

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*We are indebted to Prof. Ernest Lachman for the privilege of seeing these beautiful plates.

artery. If there are two middle lobe arteries A^6 lies at an intermediate level, i.e., in a plane drawn between the two. If there are two or more arteries to the superior segment, each may lie just below the corresponding middle lobe artery (Boyden and Hamre, Fig. 3); if there are three, the highest may lie above the middle lobe arteries, and the lowest below them (Fig. 2, #26).

THE ARTERIES OF THE SUPERIOR SEGMENT (A^6)

As summarized in Table IV, a single artery supplies the segment in 80 per cent of specimens. The prevailing pattern (50 per cent) is a bifurcation into a lateral ramus (A^6c) and into combined medial and superior rami ($A^6a + A^6b$, Plate 2). This, it will be recalled, is also the prevailing pattern of the bronchi (72 per cent, Table II), but an analysis of all specimens indicates that a given arterial pattern reproduces the bronchial pattern in only 56 per cent. In other words the arterial rami cross the subsegmental planes in 44 per cent of specimens. Sometimes the arteries trifurcate, or are branched like a shrub or merely have another type of bifurcation.

In 16 per cent there are two separate arteries (cf. #17, Fig. 2) and in 4 per cent three separate arteries (cf. #26, Fig. 2). In sum, more than one artery is to be looked for in one-fifth of the lobes. Preoperatively, these could not be predicted except in those cases in which two orifices for B^6 have been demonstrated by bronchography or at bronchoscopy. (See discussion following Table II.)

Another impediment to the resection of this segment is the fact that in 10 per cent of specimens A^6 carries a displaced ascending artery to the upper lobe. Almost always this supplies the posterior segment (cf. A^2b, Fig. 2, #26).

In concluding this section, attention should be called again to the importance of the interlobar part of the right pulmonary artery in segmental resection and lobectomy (cf. Lindskog, Liebow and Hales; Boyden and Hamre).

TABLE IV. ARTERIAL SUPPLY OF SUPERIOR SEGMENT

I. Segment supplied by *single* artery bifurcating into:			80%
1. Simple types		64%	
1) A^6c and $A^6a + b$ (Plate 2)	50%		
2) A^6a and $A^6b + c$ (Fig. 2, #48)	8%		
3) A^6b and $A^6a + c$	6%		
2. Complex types (with displaced branches)*		16%	
II. Segment supplied by *two* separate arteries (Fig. 2, #17)			16%
1. Simple types (A^6a or A^6c separate from the rest)		8%	
2. Complex types		8%	
III. Segment supplied by *three* separate arteries (Fig. 2, #26) A^6c split, or A^6b split			4%

*For example, $A^6b + a1$ and $A^6c + a2$.

THE ARTERIES TO THE MEDIAL AND ANTERIOR BASAL SEGMENTS (A^7, A^8)

In seeking a method by which the branches of the basal portion of the pulmonary artery may be classified it has been necessary to eliminate the "dorsal" rami (subsuperior arteries), since they are as variable in origin as the corresponding bronchi (B^*, BX^*). When these are ignored, the distribution of arteries to the medial basal and anterior basal segments (B^7 and B^8) seems to be correlated with the bronchial patterns shown in Plate 4. In the 56 per cent of specimens which makes up Types I and II (the normal pattern) the first anterior branch of the basal artery is either the medial basal (38 per cent; cf. #48, Fig. 2) or A^{7+8} (18 per cent; cf. #17, Fig. 2).[*] In the remaining 44 per cent of specimens (Types III and IV, in which B^7 either surrounds the basal veins or is absent as such) all but three of the specimens (6 per cent) are characterized by a displacement of the medial ramus (A^7b). That is, the latter shifts posteriorly, either to arise from the basal artery or to become an accessory branch of A^*, AX^* or A^{10} (AX^7b, Plate 5, #24). Meanwhile A^7a remains in its usual anterior position (A^7a, Plate 5, #1) or shifts laterally to become an accessory branch of A^8 or A^9 (AX^7a, Plate 5, #24).

Another striking feature is the variability of the artery to the anterior basal segment (A^8). Smith and Boyden showed that B^8 was the most constant of the basal bronchi of the right lung, and this has been verified in our series; for in 94 per cent of specimens it arises as a single branch of the stem bronchus.[†] Yet the corresponding artery (A^8) arises as a single branch of the basal artery in only 48 per cent of specimens. In another 16 per cent (12 and 4) it is associated with either A^7 or A^7a (cf. Plate 5, #1); in another 12 per cent it is associated with A^9 (cf. Plate 5, #24). In the remaining 24 per cent it is split, arising as two separate arteries from the basal trunk (Plate 3) or as two separate arteries associated with portions of A^7 or A^9, or with the whole of A^9.

Perhaps the variability of A^8 may be explained by the fact that it lies next to a variable segment, but this does not explain the numerous variations encountered in other basal arteries (vide infra). In any event the importance of these arterial anomalies of A^8 are obvious since hitherto the anterior basal segment has been considered one of the easily resectable segments.

THE ARTERIES TO THE LATERAL AND POSTERIOR BASAL SEGMENTS (B^9, B^{10})

The basal portion of the right pulmonary bronchus may be considered to terminate in a bifurcation—namely, in the lateral basal and posterior basal segmental bronchi. In the fifty specimens studied by us, B^9 was absent as such—that is it developed as an accessory branch of B^8—in only 4 per cent. (Smith and Boyden found it in 8 per cent.) Similarly, in our study there was only 10 per cent of specimens in which B^9b arose from B^{10}, or $B^{10}a$ from B^9b.[‡]

Accordingly, one would expect that with relatively so few variations in the forking of the bronchi (14 per cent) there would be correspondingly few variations in the forking of the arteries. Strangely enough, however, the basal artery terminates normally by bifurcating into segmental arteries A^9 and A^{10} in only 44 per cent of specimens (cf. Plate 3).

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[*] In three specimens there were minor variations: in one, the basal artery trifurcated into A^7, A^8, A^{9+10}; in the other two, A^8a (or a part of it, Fig. 2, #26) branched off lower down (Plate 3).

[†] In the remaining specimens (6%), B^8a and B^8b had separate origins of varying sorts (cf. Fig. 1, #26).

[‡] There are other variations which do not affect the arrangement of arteries, such as an absence of B^9a in 16% and the presence of a high paravertebral branch of B^{10} in 14%.

In the remainder many types of variations occur. Thus in 18 per cent A^9 is split, its two rami arising separately from the basal artery or in common with branches to other segments. In 30 per cent, A^9 is atypically associated with A^8, A^{7-8}, A^* or AX^*; and in 8 per cent the branches of A^9 and A^{10} are mixed.

THE ARTERIES OF THE SUBSUPERIOR ZONES (A^* AND AX^*)

A summary of the occurrence and origin of the subsuperior and accessory subsuperior bronchi in 100 specimens (from our data and those of Smith and Boyden) is presented in Table V. It will be recalled that B^* corresponds to the second dorsal ramus of Aeby, BX^* to the third and B^6 to the first. (See succession of dorsal rami ending in $D4$ in Plate 7.)

Forty-two arteries to 36 subsuperior bronchi (B^*) occur in thirty of our fifty specimens. In exactly four-fifths of these the arteries arise on the dorsal side of the basal artery either high up (51 per cent), in the vicinity of A^7 or A^8 (Plate 6, #5A), or lower down (29 per cent), nearer the bifurcation of the basal artery into A^9 and A^{10} (Plate 5, #1). The remainder arise aberrantly from A^{10} (10 per cent) or A^9 (10 per cent). The larger number of arteries is due to the fact that the arterial rami which accompany a given subsuperior bronchus may arise from more than one site. Because of the deep posterior location and the variable origins of these arteries, resection of a subsuperior segment would seem to be almost impossible unless the superior segment (B^6) had been removed first.

TABLE V. SUBSUPERIOR BRONCHI (B^* AND BX^*)

I. Frequency of occurrence in 100 specimens			
1. Subsuperiors arise exclusively from level d_2 of stem bronchus as B^*			16%
2. Subsuperiors arise exclusively from level d_3 as BX^* from B^{10}			39%
3. Subsuperiors arise from both levels in same specimen			45%
			100
II. Detailed analysis			
1. Subsuperior represented by a single bronchus		43%	
(1.) Arising at d_2 level, as B^*	13%		
(2.) Arising at d_3 level, as BX^* (10)	30%		
2. Subsuperior represented by two bronchi		43%	
(1.) Two B^*	3%		
(2.) One B^* and one BX^* (10)	32%		
(3.) Two BX^* (10)	8%		
3. Subsuperior represented by three bronchi		13%	
(1.) Two B^* and one BX^*	8%		
(2.) One B^* and two BX^*	4%		
(3.) Three BX^*	1%		
4. Subsuperior represented by four bronchi			
(1.) Two B^* and two BX^* (10)		1%	
III. Origin of B^* (at d_2 level). (Items I:1 and I:3)			61%
1. One subsuperior		48%	
(1.) At level of B^7	10%		
(2.) Between B^7 and B^8	18%		
(3.) Between B^8 and B^9	20%		
2. Two subsuperiors		13%	
(1.) At level of B^7 and between B^8 and B^9	2%		
(2.) Between B^7 and B^8 and between B^8 and B^9	5%		
(3.) Between B^8 and B^9	6%		

Fifty-eight arteries to 52 accessory subsuperior bronchi *(BX^*)* occur in forty-one of our fifty specimens. Nearly seven-eighths of these (86 per cent) arise from A^{10}, 9 per cent from the lower part of the basal artery, and 5 per cent from A^9. Thus, as might be expected, the subsuperior arteries originate prevailingly from the upper half of the basal artery while the accessory subsuperiors arise chiefly from A^{10}.

Returning to the bronchi, only one correlation can be made between B^* and the four patterns presented in Plate 4, but this is very striking. In Type III, B^* is present in only 42 per cent of specimens of that group, whereas in Types I, II and IV the respective percentages are 59, 82, and 60. From these figures it would appear that when B^7b lies on the deep side of the basal vein (Type III) there is the least room for subsuperiors to develop and when B^7 lies most anterior (Type II) there is most room.

Another interesting comparison can be made between the position of the highest BX^* (the high posterior branch of B^{10}) and B^9a, the high posterior-lateral branch of B^9. The keel of each lies about a centimeter below the keel separating B^9 from B^{10} (Fig. 1, #17 and #37), but that of BX^* is slightly higher, averaging 7.2 mm., with a range of from 2 to 15 mm. (thirty-eight specimens) while that of B^9a averages 11 mm. with a range of from 7 to 14 mm. (thirty-nine specimens).*

CONCLUSIONS

Analysis of the segmental bronchi, veins, and arteries in fifty dissected specimens of the right lower lobe has led to the conclusion that the most important relation in the basal half of the lobe is that between the bronchi of the medial basal segment and the large venous trunks which enter the inferior pulmonary vein.

The inferior pulmonary is formed by a confluence of the vein from the superior segment *(V^6)* and the common basal vein. The latter is formed by the union of superior and inferior basal veins that drain the basal segments. The upper basal comes largely from segments 8 and 9, the lower basal chiefly from segments 9 and 10.

The mode of formation of the inferior pulmonary vein is anomalous in 22 per cent of specimens. It may be formed by three or four trunks and receive major tributaries from the middle and even the superior lobe.

Four types of the medial basal segment *(B^7)* are recognized: (I), the prevailing pattern, that in which the segmental bronchus *(B^7)* is distributed to both anterior and paravertebral surfaces, and passes superficial to the basal veins (34 per cent); (II) a slight modification of the prevailing pattern in which the distribution of B^7 is limited to the anterior (and diaphragmatic) surfaces (22 per cent); (III), a major variation in which B^7 divides around the common basal vein (24 per cent); (IV), a major variation in which B^7 is absent as such (20 per cent), B^7a arising as an accessory branch of the anterior basal bronchus *(B^8)*, and B^7b arising as an accessory branch of the subsuperior *(B^*)* or the posterior basal bronchus *(B^{10})*.

Resection of Type III is considered to be hazardous, and criteria are presented by which it can be recognized preoperatively. Resection of Types I and II are feasible, and a description is presented of the intersegmental veins along which the segment may be resected.

Similarly, attention is called to the intersegmental veins which separate the superior *(B^6)* from the basal segments *(B^{7-10})*. The arteries to the superior segment are multiple in one-fifth of specimens. In 8 per cent the bronchi of the superior segment arise from two separate orifices 6 to 10 mm. apart; thus the lower B^6 might be confused with a high subsuperior bronchus *(B^*)*.

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*In additon to the thirty-nine there are three specimens in which B^9a arises atypically at the orifice of B^9. In the remaining eight it is absent.

The basal portion (pars basalis) of the inferior pulmonary artery lies on the posterolateral side of the stem bronchus. In 44 per cent, it terminates by forking into A^{9} and A^{10}, the arteries of the lateral basal and posterior basal segments. In the remainder, the lower arteries arise irregularly. The first anterior branch of the basal artery is A^{7} (or A^{7+8}) in Types I and II (56 per cent) but in Types III and IV (44 per cent) $A^{7}b$ is nearly always posterior in origin. A^{8} arises as a single branch of the basal artery in only 48 per cent. In the remainder it is split in origin (24 per cent) or arises in conjunction with A^{7} or A^{9} (28 per cent). The situation is further complicated by the variable origin of multiple arteries to the subsuperior zones (100 in fifty specimens).

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LOBECTOMY FOR FUNGOUS ABSCESS OF THE LUNG: EFFECT OF PENICILLIN

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THERE is a good deal of evidence to show that the administration of penicillin encourages fungous infections, and it is probably for this reason that these infections are on the increase. The following case is of interest because:

(1) A huge pulmonary abscess was treated with penicillin which eradicated all organisms except a fungus.

(2) Heroic doses of penicillin nevertheless kept the clinical symptoms under control while the patient was being prepared for surgery.

(3) The patient survived lobectomy in spite of his age and desperate condition.

CASE REPORT

The patient was a 72-year-old, American-born grocer who was first observed by one of us (M.M.) in October, 1949. Presenting symptoms were cough, hemoptysis, weakness, fever, and nosebleeds. He had been in good health until 1947 when a chest film showed a density in the right apex. He started going downhill, but he continued active until September, 1949, when the above-mentioned symptoms of lung abscess developed. Sputum was mucopurulent, occasionally bloody. Fever went as high as 102° F.

Physical examination showed that the man was critically ill. He was dyspneic, cyanotic, toxic, and emaciated. His blood pressure was 122/74; pulse was 80. There was a maximal precordial impulse in the fifth intercostal space at the midclavicular line. A low, blowing, musical, systolic murmur replaced all heart sounds, and was transmitted over the entire upper chest. Rhythm was regular. Showers of râles were present at the right apex. Prostate was enlarged, grade I. Diffuse, patchy, scaly eruption over the sternum was noted.

A chest film revealed a giant cavity in the right upper lobe, measuring 4.7 cm. by 7 cm. (Fig. 1). There was no fluid level, but the lower three-fourths of the cavity contained a "cotton ball" density. A lateral view showed the cavity to be occupying the posterior segment of the upper lobe. The interlobar fissure was shifted upward, and the trachea was pulled to the right. The remainder of the lung showed fibrosis and emphysema. The heart was normal in size. An electrocardiogram revealed T-wave and ST-segment changes indicative of myocardial disease.

Laboratory work showed hypochromic anemia, hypoproteinemia, and mild azotemia. There was a persistent eosinophilia varying from 5 to 21 per cent. Sputum cultures at first showed a monilia-like fungus. Later they became sterile. Bronchoscopy showed purulent material coming from the right upper lobe bronchus. This material failed to show a fungus on culture. A needle biopsy somewhat later in the course of the illness showed a great abundance of septate mycelium in necrotic lung tissue. Morphologically it resembled Aspergillus; culturally it was sterile. Intradermal tests for cococcidioidin and histoplasmin were negative in twenty minutes and forty-eight hours. Oidiomycin showed a two plus reaction in twenty minutes with no delayed reaction. An autogenous vaccine of the monilia-like fungus from the initial sputum culture showed a two plus reaction on intradermal tests in twenty minutes.

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Course.—Therapy was directed towards combating the pulmonary infection and overcoming the toxemia. The patient was treated with intravenous amino acids, blood transfusions, crude liver extract, and vitamins. Digitalis was given from the start. Parenteral penicillin was started with an initial dose of 1 million units a day. The temperature which at first ranged as high as 104° F. returned to normal, and the symptoms improved. On November 11 the penicillin was cut to 240,000 units a day. In a few hours the patient showed an increase in toxicity with a rise in temperature. Because of this, the penicillin was increased to 500,000 units per day and then gradually to 1 million, 3 million, 8 million, and finally 20 million units a day. The cavity shrank noticeably (Fig. 2). Three times an attempt was made to reduce the dose, but each time the patient reacted with signs of increased toxicity. On two of these occasions he suffered massive hemoptysis. Aureomycin, chloromycetin, gantrisin, dihydrostreptomycin, and iodides were all tried but without apparent benefit.

The patient now developed urinary obstruction. Because his condition was still too poor for surgery, he was placed on catheter drainage, and after several weeks he had a transurethral prostatectomy. He recovered. A few days later the needle biopsy of the lung was repeated. The findings were the same as at first.

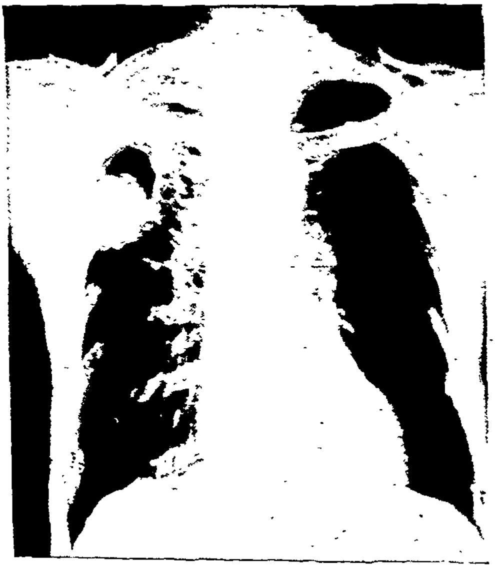

Fig. 1.—Appearance of the lesion when the patient was first seen.

Surgery for the pulmonary lesion was undertaken on March 3, 1950 (C.L.G.). At thoracotomy the right upper lobe was found densely adherent to the parietal pleura. The posterior segment of the upper lobe was heavily infiltrated, and this process also involved the superior segment of the lower lobe. Because of the extent of the disease, and because carcinoma was still not entirely ruled out, a lobectomy was decided upon. The hilum was surprisingly free. The branches of the pulmonary artery and of the superior pulmonary vein were individually ligated and divided. The bronchial stump was closed with 00 silk. A small amount of the superior segment of the lower lobe was also removed. The wound was closed, and two catheters were inserted into the chest, one in the posterior axillary line, and one in the midclavicular line. Through these catheters penicillin and streptomycin were inserted into the pleural cavity during the first two postoperative days.

The patient did poorly at first because of severe bronchial asthma and cerebral anoxia. He was confused, agitated, and disoriented. These symptoms gradually subsided. The lung expanded well, except for a small pocket at the apex which has persisted to date. Vital capacity has remained practically unchanged. The patient went home on the twelfth postoperative day and has remained well (Fig. 3).

Examination of the operative specimen showed a chronic lung abscess 5 cm. in diameter, draining into the posterior division of the right upper lobe bronchus. It was filled with a reddish-black, necrotic slough. Microscopic examination showed that the walls were covered with the same type of fungus as had been noted in the needle biopsies. Cultures were again sterile.

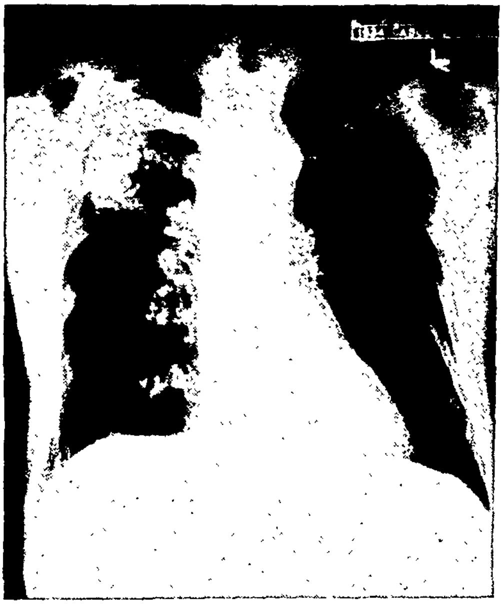

Fig. 2.—After three months of penicillin therapy.

COMMENT

In summary, this was an elderly patient who developed a pulmonary lesion of undetermined nature in 1947. Two years later this lesion rapidly broke down and formed a giant abscess which was partially controlled with tremendous doses of penicillin. Cultures were negative except at the very beginning (before penicillin was started). Two needle biopsies and the operative specimen showed a fungus, either Candida or Aspergillus. After a stormy preoperative course the patient came to lobectomy. He survived, and is now as well as ever.

There are several interesting features in this case. In the first place, what was the role of penicillin? Did it enhance the growth of the fungus, or did it simply suppress the normal competitors? Secondly, why did penicillin produce clinical improvement and shrinkage of the cavity? Thirdly, this patient illustrates that lobectomy for lung abscess is not necessarily contraindicated in the presence of old age, general debility, advanced arteriosclerosis, and severe cardiac damage.

The effect of penicillin on fungi has been made the subject of several investigations and a number of case reports. As early as 1942 Hobby, Meyer, and Chaffee[1] tested various strains of fungi for penicillin-sensitivity in the test tube and found that all were penicillin-resistant with the exception of Cryptococcus hominis. In 1944 Keeney, Ajello, and Lankford[2] found that *Candida albicans, Cryptococcus neoformans, Histoplasma capsulatum,* and *Coccidioides immitis* were not only unaffected, but actually stimulated by penicillin in the test tube. In 1946 Fleming and Queen[3] repeated these experiments with nineteen different strains of fungi. Their conclusions were that all were penicillin-resistant, with the exception of Actinomyces (there is also good clinical evidence that actinomycosis is favorably influenced by penicillin[4]). In 1947 Mattman[5] reported that the common fungi were consistently resistant to penicillin and to streptomycin in laboratory tests. In 1949 Foley and Winter[6] noted that throat cultures from patients treated with penicillin for diphtheria and scarlet fever often showed an abnormal preponderance of fungi. They then injected various fungi into chick embryos and observed that the mortality was greatly increased by the addition of penicillin. They thought that penicillin had a direct stimulating effect on the fungus, rather than a suppressing effect on competitive organisms.

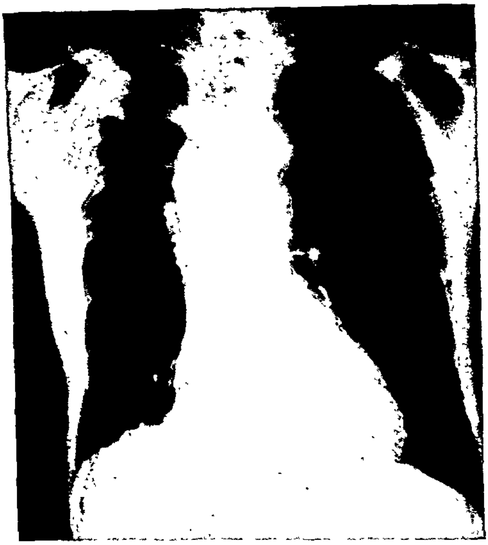

Fig. 3.—Postoperative appearance of the chest.

Besides these experimental observations, there are several case reports suggesting that penicillin makes the patient susceptible to fungi. In 1946 Geiger, Weimer, Axilrod, and Durlacher[7] described a patient who developed a fatal *Candida albicans* endocarditis and meningitis during intensive penicillin therapy for subacute bacterial endocarditis. In 1950 Grekin, Cawley, and Zheutlin[8] reported fatal pulmonary aspergillosis in a patient who was under penicillin treatment for influenza. In the same year Zimmerman[9] reported three cases of Candida and Aspergillus endocarditis, all of whom had been under treatment with penicillin and various antibiotics. He felt that there was an etiological connection and quoted as yet unpublished work by Farber pointing in the same expanded well, except for a small pocket at the apex which has persisted to date. Vital capacity has remained practically unchanged. The patient went home on the twelfth postoperative day and has remained well (Fig. 3).

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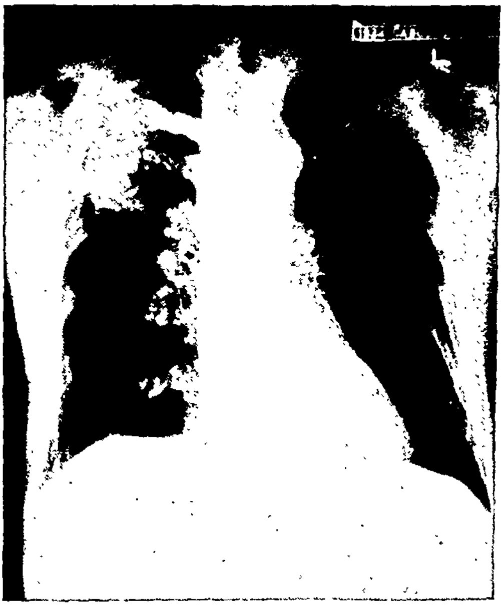

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SUMMARY

A man 72 years of age who was severely ill with a huge pulmonary abscess that showed only fungus organisms responded to massive doses of penicillin and survived a right upper lobectomy.

REFERENCES

1. Hobby, G. L., Meyer, K., and Chaffee, E.: Activity of Penicillin in Vitro, Proc. Soc. Exper. Biol. & Med. 50: 277, 1942.
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direction. In 1951 Woods, Manning, and Patterson[10] reported twenty-five patients who developed monilial infections while under treatment with penicillin, aureomycin, and chloramphenicol. Two of these patients had pulmonary lesions. These authors believe that the patient is made susceptible by the changes which the antibiotics produce in the normal intestinal and bronchial flora. Other authors, such as Schnurman[11] and Keeney,[2] have seen acute exacerbations of mycotic skin infections under parenteral penicillin therapy for unrelated infections. This is believed to be an allergic manifestation. The only author to report a favorable effect of penicillin on pulmonary moniliasis is Zussman,[12] but his patient received so little of the drug (a total of 1.5 million units) that it is questionable whether there was any causal relationship.

With all this evidence at hand, it seems probable that our patient had a mixed infection in which the fungi eventually became the predominating organisms. Fungi are normal inhabitants of the mouth in many people. The significant observation here is that it was possible to keep the clinical symptoms under control and even produce some shrinkage of the cavity, not by the discontinuance of the penicillin (as others have suggested) but rather by an increase in the dose to the heroic levels of 20 million units a day. Several attempts to reduce this dose led to a clinical relapse. This is difficult to explain on the basis of the known penicillin-resistance of fungi. Possibly the suppression of all other organisms had something to do with it. At any rate, it is a fact well worth recording because it was this temporary control of the infection that made operation eventually possible.

We were never able to identify the fungus in this case. Morphologically it looked like Candida or Aspergillus because of the profuse mycelium, but the morphologic characteristics of fungi are notoriously variable, and it is necessary to have cultural evidence for a positive diagnosis. However, even had we been successful in culturing this organism, the information would not have had any important bearing on the treatment. Clinically these infections are a single entity, and any variations that occur are based on fortuitous fluctuations in virulence rather than on specificity of strains.

The preferred treatment of chronic lung abscess has undergone a noticeable change in the last ten years. Whereas simple drainage used to be the treatment of choice, resection is now carried out more and more frequently. A simple drainage would never have cured this patient. There was too much destruction of pulmonary tissue. Also, there is always the question of a hidden cancer. In recent years Shaw and Paulson,[13] as well as Kent and Ashburn[14] have stressed the desirability of resection. They feel that a great many patients continue to have symptoms of residual bronchiectasis when an abscess is merely drained. The oldest patient in the series of Shaw and Paulson was 54 years of age. The oldest patient in the series of Kent and Ashburn was 64 years of age. Our patient was 72 years, but he is not the oldest patient in the literature to survive lobectomy for abscess. Rosenblatt, Aufses, and Lisa[15] have reported a successful right middle lobectomy in a patient 82 years old!

Nevertheless, the result achieved here was most gratifying. The patient was debilitated, toxic, senile, and extremely ill. He was suffering from an overwhelming infection, and nearly died of massive hemoptysis on several occasions. In addition, he had generalized arteriosclerosis, advanced pulmonary emphysema, severe cardiac damage, and long-standing prostatism. That such a patient could survive was due chiefly to the careful preoperative preparation, meticulous attention to detail during the operation. and proper supportive measures afterwards. Penicillin played an important role.

SUMMARY

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DOUBLE AORTIC ARCH

J. F. Nuboer, M.D.*
Utrecht, Holland

IN THE year 1794 Bayford applied the name of *dysphagia lusoria* to a morbid condition characterized by difficulties in swallowing and the presence of an abnormally located right subclavian artery, which passes posterior to the esophagus, causing difficult swallowing owing to its pressure. Since that date a number of anomalous developments of the aortic arch have been observed which may give rise to similar clinical symptoms. One of them is the double aortic arch, a rare irregularity; in this case the ascending aorta divides into two branches encircling the esophagus and trachea (very rarely the trachea only), uniting a little distance farther into the descending aorta.

In normal circumstances the arcus aortae develops from the left fourth branchial arch, the innominate artery and the right subclavian artery springing from the right fourth branchial arch, and the distal portion of this primitive right aortic arch disappears (Fig. 1). When development of these arteries is disturbed during the period between the third and the sixth embryonal weeks, the result may be the formation of a ring around the esophagus and trachea, consisting wholly or partly of blood vessels; this ring, if sufficiently narrow, may cause pressure on the organs within it. Sometimes the pulmonary artery and the arterial duct (both arising from the sixth branchial arch) are also involved in exerting this pressure. Examples of the latter possibility are mentioned by Gross and Ware[7] when they describe the different vascular anomalies that may be observed in this region.

According to Gross and Ware,[7] a double aortic arch in man may be observed in two shapes:

1. As a partly obliterated double aortic arch. Only one of its legs (the right one usually) is open and situated behind the esophagus; the other has become obliterated and is represented by a vestigial remnant or cord in front of the trachea. This irregularity is less common than the second.

2. As a completely open double aortic arch. The two arterial branches very rarely have the same diameter (Schall and Johnson[12]), in most cases one is wider than the other. We nearly always find a wide right arch, passing posterior to the esophagus and trachea, and becoming a descending aorta, not situated to the right of the vertebral column, yet slightly farther to the right than a normal descending aorta. The left arch is present in the shape of a slenderer arterial branch, anterior to the trachea and uniting farther toward the periphery with the right arch, so as to form the descending aorta. The great arterial vessels arising from the aortic arch, viz., innominate artery, left carotid artery, and left subclavian artery, may arise in different ways from

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the left and right aortic arches. Among the forty cases examined by Blincoe[3] there were only two where the left (anterior) arch was the wider. Similar cases were described by Gross,[6] Gordon,[5] and Potts.[10]

There are some cases in medical literature, however, where the trachea only was encircled, the posterior arch passing between the trachea and esophagus (Malacarne,[8] Sagorsky,[11] and Welch[14]).

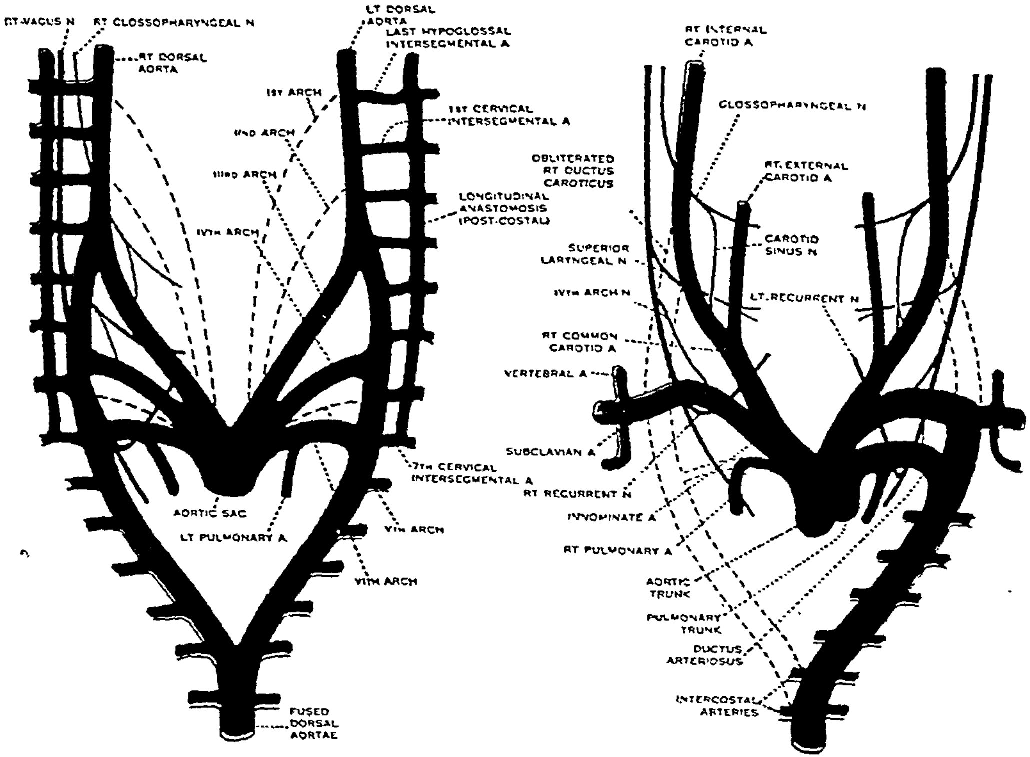

Fig. 1.—Normal development of aortic arch in man (from Hamilton, Boyd, and Mossman, *Human Embryology*).

It goes without saying that this vascular ring may compress and constrict the encircled anatomic organs. When this ring is sufficiently wide, this irregularity need not be attended with clinical symptoms; instances of this are mentioned in medical literature (Arkin[2]). Should the ring be narrow, however (and this seems to be the case in the majority of instances), the trachea and esophagus may be constricted and give rise to serious clinical symptoms. Wolman[15] has given an excellent description of them. Shortly after birth there seem to be difficulties in swallowing, and stridor is noticed, which may be inspiratory or expiratory, or both. Sometimes this symptom is observed only when the child cries, or exerts himself. At times there is cyanosis, in other cases this symptom is evoked only by crying. Intercostal and suprasternal contractions may be noticed. Moreover, the child often has fits of curious, raucous coughing. The onsets and the asphyxia are very marked during the periods of feeding, when stridor mostly increases and cyanosis develops. The action of feeding, moreover, is difficult, and takes much time, as between the acts of swallowing, the child must rest in order to recover his breath. Infection of the air passages occurs frequently; it is often caused by swallowing the wrong way, ending fatally when it is not attended to. In the seven cases collected by Wolman[15] all the patients died.

The view advocated by Apley in the Royal Society of Medicine in 1949, in a discourse entitled "Congenital Anomaly of the Aortic Arch and Its Branches" would therefore seem to be somewhat unusual, at least in the form in which it was published. In the summary it is stated that "Treatment, apart from chemotherapy of superimposed lung infections, is rarely indicated, because of the tendency to spontaneous recovery, but surgical correction may be life-saving."

It is not quite clear whether this view has special reference to a certain form of vascular anomaly in this case—the double aortic arch. If this should be so, we would observe that in those cases where the anomaly was accidentally discovered at an obduction, or where they were accidentally diagnosed in adults, there had been no clinical symptoms previously. A short time ago we found this anomaly accidentally in a man, aged 69 years, who complained of vague gastric pains. He had never before had any difficulties in swallowing, and there had not been any subjective or objective symptoms of narrowing of the trachea. On the other hand, however, we know of many cases where the symptoms arose a short time after birth, and where the patient died of pneumonia. When the irregularity reveals itself in distinct symptoms in young children, we must look upon it as very grave. Potts[10] for example, treated an 8-year-old child, who had no fewer than twenty-one times been admitted to the hospital for pneumonia before being operated upon. I therefore agree with Exalto, Dicke, and Aalsmeer,[4] when they expressed the view that in babies or young children surgical therapy is necessary when the ring of blood vessels develops clinical symptoms, in order to prevent death or repeated infections of the air passages. I would, however, add that in such a case the symptoms must be really pronounced and unmistakable, for naturally there will also be cases where the symptoms are of such a mild degree that it seems possible to carry them along without an operation. When we consider the fact that Gross and Neuhauser[16] report four deaths in sixteen cases, it seems necessary to avoid the greater risk of an operation in such mild cases, where the anomaly itself will mean a very slight risk for the patient.

With the aid of a roentgen examination, a diagnosis can be made during life. Neuhauser[9] pointed out that after swallowing barium a filling defect may be observed in the posterior wall of the esophagus due to pressure of the right posterior aortic arch. The trachea proves to be constricted at the same place, which may be made visible in an ordinary roentgen photograph; if required, lipiodol may be introduced into the air passages.

It is hardly necessary to say that the only rational therapy of this irregularity consists of cutting the constricting vascular ring. The operation in itself is not difficult. As far as I have been able to ascertain it has up to now been successfully done on eighteen patients only.

In 1945 Gross[6, 7] published the first successful case, in 1946 he published a communication about a second patient cured, and from a correspondence with Potts[10] it appears that he had a third successful case, which had not yet been published. In 1947 Sweet[13] mentions two patients who were successfully operated on. In 1948 Potts[10] described a female patient who was operated on with complete success, and he also mentioned the case of another child where the cause of the constriction could not be found during the operation, as the constricting vessel lay hidden behind the esophagus; this child died some days later of suffocation. In 1950, finally, Exalto, Dicke, and Aalsmeer[4] published three cases of the same anomaly which were treated successfully.

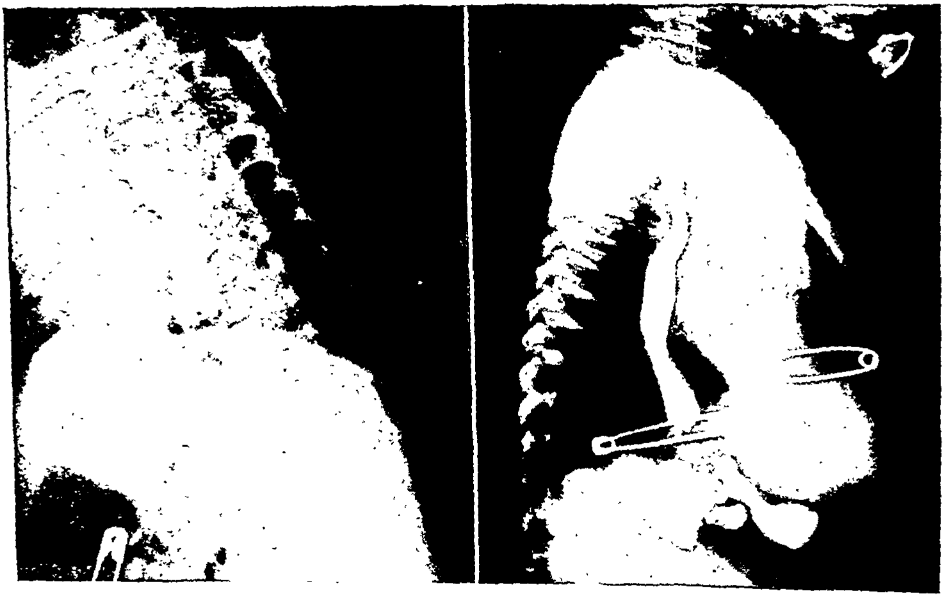

Fig. 2. Fig. 3.

Figs. 2 and 3.—Roentgenogram of esophagus after barium filling. A filling defect will be observed toward the rear, at the level of the third and fourth thoracic vertebrae.

In *Pediatrics* (January, 1951) Gross and Neuhauser[16] reported on no fewer than sixteen cases of double aortic arch which were surgically treated. The ages varied between 1 month and 2 years. In thirteen patients there was a left descending aorta; in nine of them the left (anterior) arch was the smaller of the two and was divided; in four cases the smaller right (posterior) arch was cut. In the three cases where a right descending aorta was seen, it was always the left (posterior) arch that was divided. Four of these sixteen patients died. The cases published earlier by Gross are included in this series.

I can add the following case to this short series:

A 3-month-old Belgian boy from Antwerp, born Aug. 12, 1950, the fourth child of healthy parents (weight at birth 4,000 Gm.), was admitted to the Surgical Department of the University Hospital at Utrecht on Nov. 23, 1950. From his birth onward it was noticed that the child suffered from a distinct stridor. Supraclavicular and intercostal inspiratory contractions were observed. The child cried a great deal, and he often had mucus in the air passages, causing fits of coughing. Respiration was easier when he was held erect. The act of drinking caused troubles from the first; the child drank very slowly, feeding had to be interrupted repeatedly, and he easily choked when swallowing. When he was crying, cyanosis developed. The pediatrician in Antwerp, Dr. Helderweirt, made roentgen photographs, which at first revealed only hypertrophy of the thymus. After the esophagus had been filled with barium, a considerable depression in the posterior wall of the esophagus was observed near the third and fourth thoracic vertebrae (Figs. 2 and 3); in the anteroposterior roentgenograms, too, a constriction could be seen at this site. The trachea also proved to be narrowed here (Fig. 4). An attempt to fill the trachea with lipiodol failed because it caused an onset of extreme suffocation. A diagnosis of ''double aortic arch'' having been made, the child was sent to us for surgical treatment.

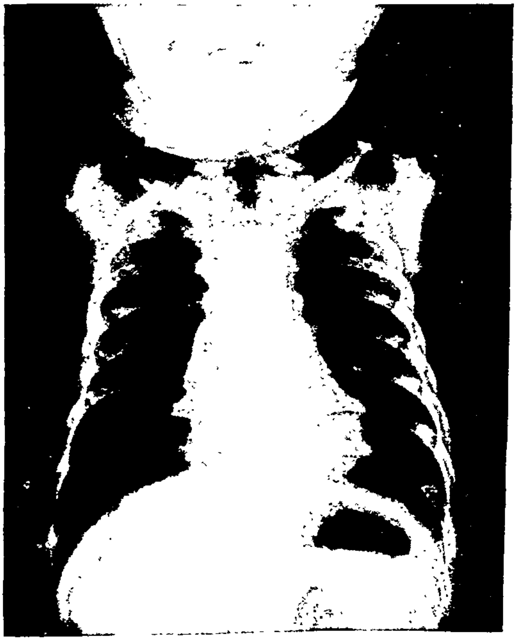

Fig. 4.—Roentgenogram of trachea. A stricture is visible just above the bifurcation.

On repeating the roentgenologic examination the above findings were fully confirmed. Considering the circumstance that symptoms of pressure on the trachea and esophagus could be observed, which are characteristic of constriction by a double aortic arch, the diagnosis seemed to be correct. The child showed symptoms of serious constriction of the two anatomic organs, and therefore the prognosis without surgical treatment was very bad; hence it was decided to proceed to an operation. The patient was somewhat more than three months old, its weight was 5,700 Gm., and its general condition was good.

The operation was performed on Dec. 4, 1950, intratracheal anesthesia of cyclopropane-oxygen being administered. The tracheal catheter number 1 could not be advanced beyond the stricture, and up to the moment when the constricting ring was cut the anesthetist found it very difficult to make the gases pass the site of the stricture in the air passages. The patient lay on his right side, and the thoracic cavity was opened in the third intercostal space. The large thymus was at once conspicuous. There was no aortic arch on the left side, and at first the descending aorta could not be seen. Below the pleura a fairly large pulsating vessel was visible, which immediately gave the impression of being the patent ductus Botalli. The mediastinal pleura was incised between the phrenic nerve and the vagus nerve, exposing the left pulmonary artery. The blood vessel observed earlier proved to be the long ductus Botalli. After the thymus was moved aside the anatomic situation became clear (Fig. 5).

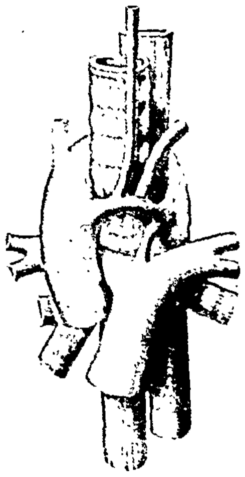

Fig. 5.

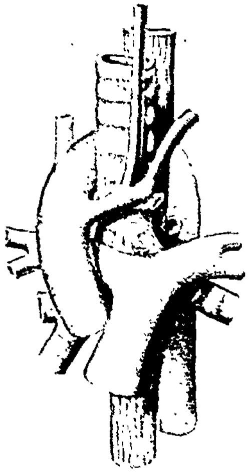

Fig. 6.

Fig. 5.—Anatomic situation as revealed by the operation. There is a wide, right, posterior aortic arch, and a narrow, left, anterior arch, converging left and right behind the esophagus, forming the descending aorta, which lies somewhat farther to the right than normally. At the site where the anterior and posterior arches unite the former is slightly widened, and receives the long ductus Botalli. From the anterior arch rise the left carotid and subclavian artery; the innominate artery could not be identified, it probably sprang from the posterior arch. Esophagus and trachea are constricted by a ring of blood vessels.

Fig. 6.—Anatomic situation at the end of the operation. The ductus Botalli has been resected, the anterior aortic arch has been ligated and divided at the point of union with the posterior arch.

There appeared to be a fairly slender anterior aortic arch, from which the left subclavian artery and the left carotid arose. The posterior aortic arch was short and thick. It passed into a descending aorta, which lay slightly farther to the right than is normal, exactly in front of the vertebral column. Near the site where the anterior and the posterior aortic arch united, slightly to the left and behind the esophagus, the anterior aortic arch was somewhat wider and received the ductus Botalli. The right carotid and the subclavian artery could not be identified; these vessels probably arose on the right from the posterior aortic arch. We could see very clearly that the trachea and esophagus were constricted by this ring of blood vessels.

When the anatomic situation had become clear, the type of operation suggested itself speedily. A double ligature was applied to the ductus Botalli, and the adjacent part then resected. Next a double ligature was also applied to the anterior aortic arch at the site of the union with the posterior arch, followed by resection (Fig. 6). At that moment the esophagus and the trachea were released from their stricture. The esophagus protruded in a lateral sense, and at the same time the trachea became passable so that the catheter could be advanced beyond the stenosis. The thoracic cavity was closed again without drainage.

After the operation the child was placed in an oxygen tent. His condition was excellent, although a slight stridor remained. Feeding then involved no further difficulties. The oxygen tent was removed four days later, recovery was uneventful, and on the fourteenth day the patient was discharged. Before his departure fresh roentgenograms were made. The site of the former stenosis in the esophagus was then readily passed by the barium meal (Figs. 7 and 8).

Reports from the parents and the pediatrician at Antwerp show that the child is doing very well, and that the stridor has gradually disappeared.

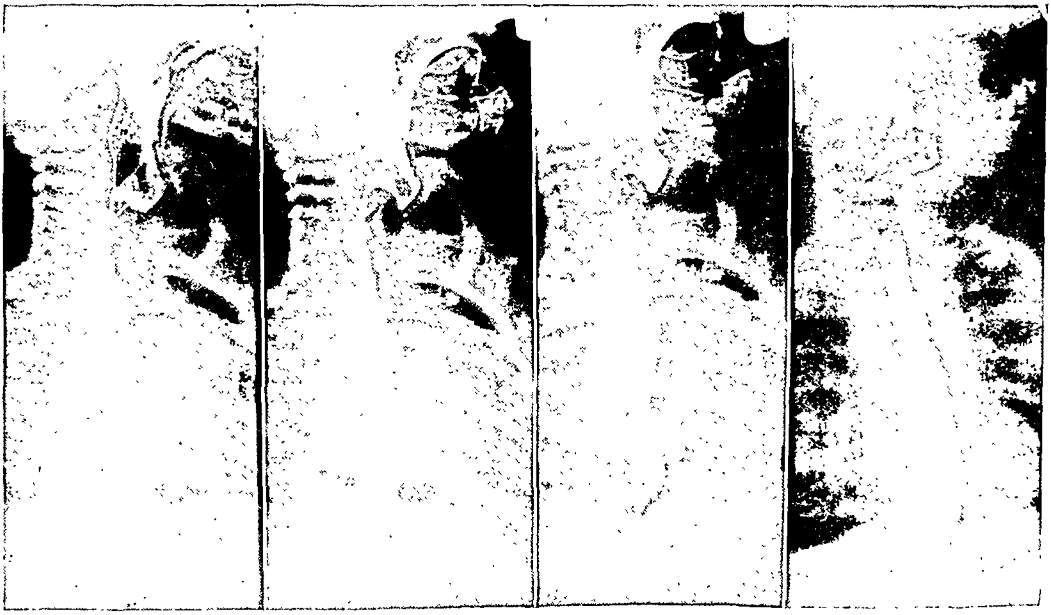

Fig. 7. Fig. 8.

Fig.. 7 and 8.—Roentgenograms of the esophagus after the operation. The stricture is no longer visible, and the barium meal passes very easily.

Exalto et al[4] point out that although the patient's condition, generally speaking, immediately shows a considerable improvement, yet there remained a slight stridor for some time in the patients they treated; it diminished slowly during the subsequent weeks, and finally vanished completely. This agrees with our experience. It would seem quite reasonable to assume (with these authors) that the stridor is caused by a softening and distortion of the cartilage rings of the trachea, which have been constricted from the third embryonal week. These irregularities will only disappear gradually.

SUMMARY

A report is given of a case of congenital constriction of trachea and esophagus, caused by a compressing double aortic arch in a child aged a little over 3 months. An operation was successful.

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X-RAY THERAPY IN CARCINOMA OF THE ESOPHAGUS

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CARCINOMA of the esophagus may be treated either by surgery or irradiation, the five-year cure rate from each being extremely poor. Surgery carries with it a considerable immediate mortality, even under the best conditions. It promises little more than the removal of the primary, and is usually of a mutilating nature. True carcinoma of the esophagus is almost invariably of an epidermoid type, and as such has a moderate radiosensitivity. Radiotherapy of this disease has been abandoned in many centers in favor of surgery, or at least relegated into a place of secondary importance due to the recent technical improvements in thoracic surgery and the advent of the antibiotics.

It is the purpose of this paper to show that cancer of the esophagus can be eradicated with properly planned deep x-ray therapy, at least in the primary site, and an almost normally functioning esophagus restored.

We wish to present four cases of carcinoma of the esophagus, all proved by biopsy, and treated by multifield beam-directed deep x-ray therapy,[1] on whom postmortem examination revealed no residual tumor in the esophagus or in the near-by tissue.

From January, 1946, to December, 1950, nineteen cases of cancer of the esophagus were seen. Six patients were not treated, owing to the presence of distant metastases or poor general condition. All of these patients died within four months. Another patient refused any treatment. Twelve patients were treated with deep x-ray therapy. Five of these patients, all proved epidermoid carcinomas, are living and apparently free from disease locally for periods up to fifty-two months. The remaining seven died from three to thirty-eight months after treatment.

Of the seven treated patients who died, autopsies were obtained on only four. Biopsies before treatment in each of these cases showed epidermoid carcinoma. In each autopsy the esophagus, and the surrounding tissue were carefully examined, and multiple microscopic sections were taken. No evidence of carcinoma was found in the treated area.

CASE REPORTS

CASE 1.—E. K., a man aged 66 years, was admitted to the hospital in August, 1947, complaining of increasing difficulty in swallowing solids for two years. He was still able to eat very soft solids by following each mouthful with fluids. No weight loss was evident. Roentgenogram examination revealed irregularity in the lower esophagus, but the barium

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From the Saskatoon Cancer Clinic.

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passed into the stomach. Esophagoscopy showed a friable ulcer on the anterior wall two inches below the aortic arch, and one inch further down it extended around the lumen to involve the posterior wall. The scope was passed to the cardia, and for one inch above the cardia the mucosa was normal. Biopsy was epidermoid carcinoma, grade II. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma, F.S.D., 80 cm., H.V.L. 4.4 mm. Cu., six fields each 15 by 6 cm. delivering a total tumor dose to the esophagus of 6,000 r in five weeks. There were twenty-five treatments, each field daily.) Barium swallow in December, 1947, revealed a smooth esophagus with slight narrowing. He was able to swallow all foods easily. In June, 1948, it again became difficult to get solids down. Roentgenograms showed no change, and on esophagoscopy no evidence of cancer could be found. Dilatation with esophageal bougies was done intermittently. Swallowing improved, and he remained well until August, 1950, when he was readmitted to the hospital. On examination the liver was enlarged, there was a large mass in the epigastrium, and a weight loss of twenty-one pounds. Roentgenograms showed that the lower end of the esophagus was deviated far to the left by a mass outside the esophagus and stomach. He died three years and two months after the treatment. At autopsy no evidence of cancer was found in the esophagus. The scarred area, where the tumor had been situated, was completely covered with epithelium, and no cancer was seen in the numerous microscopic sections. There was metastatic involvement of the gastro-hepatic and peri-aortic lymph nodes, and from hence invasion of the stomach wall. Liver, pancreas, and thirteen vertebrae were replaced by cancer.

Case 2.—J. R., a man aged 60 years, was admitted to the hospital in October, 1947, with difficulty in swallowing everything except semi-solids and fluids for one month, and ten pounds weight loss. Roentgenogram of the chest showed a focus of old inactive tuberculosis in the left apex. On barium swallow an irregular narrowing was seen in the mid-esophagus, and on esophagoscopy a fungating ulcerated mass was seen in the middle third extending onto the right wall and the whole of the posterior wall for a length of two inches. The mass appeared to be deep in the tissue behind the esophagus and fixed. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma. F.S.D. 80 cm., H.V.L. 4.4 mm. Cu. through six fields, each 6 by 15 cm., delivering a tumor dose to the esophagus of 5500r in three weeks. Each field was treated daily for fifteen treatments.) The tuberculous focus in the left apex became active after the treatment, and the patient died in the Sanatorium four months later. Autopsy revealed no residual cancer in the esophagus on multiple microscopic sections. The treated area had not yet completely re-epithelized. Bilateral fibrocaseous tuberculosis was present with cavitation in the left apex, and there was terminal pulmonary embolism. No metastatic cancer was found. It is well known that x-ray therapy will activate pulmonary tuberculosis, but in this case it was considered that the risk was justified.

Case 3.—N. S., a man aged 63 years, was admitted in November, 1947, with pain in the retrosternal area and difficulty in swallowing food, and food sticking in the lower esophagus for four months. No weight loss. Roentgenogram revealed an irregularity in the mucosal pattern of the lower five inches of the esophagus with normal mucosa seen below the diaphragm. On esophagoscopy a fungating growth with narrowing involved the anterior and right side of the lumen in the lower five inches of the esophagus. It was impossible to pass the scope beyond the lower edge of the growth. Biopsy was epidermoid carcinoma, grade III. The patient was treated with beam-directed deep x-ray therapy. (Four hundred kv., 5 Ma, F.S.D. 80 cm., H.V.L. 4.4 mm. Cu., through six fields each 15 by 6 cm., delivering a tumor dose of 5500r in three weeks. Each field was treated daily for fifteen treatments.) The patient remained well and was able to eat all foods until April, 1948, when he developed retrosternal pain related to exertion. In December, 1948, he was admitted to the hospital in congestive failure, and did not respond to treatment. He died fifteen months after treatment. Autopsy revealed no evidence of cancer in the esophagus, although multiple microscopic sections were done, and no evidence of distant metastases was present. The patient died with congestive failure incident to cardiac infarction following coronary thrombosis.

Case 4.—J. T., a man aged 52 years, was admitted January, 1950, complaining of increasing difficulty in swallowing solids for eight months and taking liquids only. He had a rib resection for empyema and a pericarditis in 1918, but no other illness. Barium swallow showed an obstruction at the level of the aortic arch with moderate dilatation above. The upper esophagus and trachea were displaced forward by an abnormal soft tissue mass which extended up toward the neck. A barium tract one inch long was observed to extend posteriorly from the esophagus into the mass. On esophagoscopy there was an extensive fungating tumor at the junction of the upper and middle thirds. It was not possible to find the lumen. Biopsy was reported as squamous cell carcinoma. The patient was given a course of beam-directed deep x-ray therapy using the Betatron (22 M.E.V., F.S.D. 105 cm., three fields, each 15 by 6 cm. delivering a tumor dose of 6000 equivalent roentgens in three weeks. Each field was treated daily for fifteen treatments.) One week after treatment was finished he had less difficulty in swallowing and barium showed the narrowing in the lumen to be less irregular. Chest roentgenograms showed the upper mediastinal mass to be smaller. The patient returned home to New Zealand and died three months later. On postmortem examination there was a bridge of normal mucosa on the anterior wall of the esophagus. The posterior wall was ulcerated and attached to the bodies of the seventh cervical and first thoracic vertebrae. The bone was exposed, and the anterior parts of the bodies formed the posterior wall of a ragged retroesophageal abscess cavity. Sections from the base and edges of the abscess were free from tumor growth. No evidence of residual cancer was found in the lymph nodes and no distant metastases.

CONCLUSIONS

It is shown that cancer of the esophagus can be eradicated by properly planned beam-directed deep x-ray therapy. Four cases with clinical history and postmortem findings are reported.

The autopsies in Cases 1, 2, and 3 were performed by Dr. J. W. Adams, Pathologist, Saskatoon City Hospital, and in Case 4 by Dr. P. P. Lynch, Pathologist, Wellington, New Zealand.

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Original Communications

CHANGES IN URINE AND SERUM ELECTROLYTES AND PLASMA VOLUMES AFTER MAJOR INTRATHORACIC OPERATIONS

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Within recent years, surgeons have become more aware of the importance of changes in fluid and electrolyte distribution and excretion following major operations. However, we have been unable to find any report of the serum concentration and renal excretion of the chief extracellular electrolytes in a series of patients subjected to major intrathoracic operations. The following study was undertaken in the hope that further information regarding the physiologic disturbances following major intrathoracic operations would result in improved pre- and postoperative care.

MATERIAL AND METHOD

Nineteen patients subjected to major intrathoracic operations were studied on the surgical service of the Jefferson Hospital. Thirteen patients had carcinoma of the lung. In seven of these, pneumonectomy was performed. Six lesions were found to be nonresectable after extensive dissection. Three patients had carcinoma of the esophagus. In two patients, an esophagectomy and esophagogastrostomy was performed. The lesion in the third patient was not resectable. In one patient a total esophagectomy and pharyngogastrostomy was performed for lye stricture of the esophagus. One patient had severe respiratory obstruction from a mediastinal tumor which was successfully removed. In the remaining case, a tuberculoma was excised. All of these operations were considered sufficiently extensive to warrant these studies.

The usual diagnostic studies were performed. Routine hospital laboratory tests gave no evidence of gross impairment of renal or hepatic function. Before operation, determinations of plasma volume and hematocrit were performed. Transfusions were given to bring the blood volume up to a level near that given as normal for the patient's surface area by the nomogram of Boothby and Sandi-

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From the Department of Surgical Research, Jefferson Medical College, Philadelphia 7.

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*Fellow, American Cancer Society.

ford.[17] The surface area was determined using the patient's height and weight prior to the onset of his illness. Blood loss was measured and replaced at the time of operation. The patients studied later in the series were given plasma or ossein gelatin* in the immediate postoperative period. Ossein gelatin prepared in 6 per cent solution is hypertonic. Five hundred milliliters have the osmotic equivalence of 750 ml. of isotonic plasma. About 50 per cent of the gelatin remains in the circulation at the end of twenty-four hours. It is largely excreted by the kidneys.[13, 24, 34] All patients were given one or two liters of 5 per cent glucose in water on the day of operation. The fifteen patients with lesions elsewhere than in the esophagus required supplementary intravenous fluids during the first or second postoperative days. Five of these patients received 76 to 153 meq. of sodium and three received 27 or 53 meq. of potassium. The four patients with esophageal disease required parenteral fluid for longer periods. Approximately 100 meq. of sodium and 26 meq. of potassium were given daily during the period of parenteral feeding. One patient considered separately below received larger quantities of sodium.

The special studies were made in the following manner in our laboratory. Blood specimens taken from an artery, or from a vein without stasis, were centrifuged within thirty minutes and the serum promptly removed. Urine specimens were collected for twenty-four hour periods. Sodium and potassium determinations were made in duplicate with the Barclay internal standard flame photometer[3, 4] which has a reproducible accuracy in our hands of 1½ per cent. The chloride determinations were done according to the method of Schales and Schales.[39] Total eosinophil counts were made promptly after the withdrawal of blood samples.[15, 21, 38, 42] Plasma volumes were measured using the T1824 dye dilution technique of Gibson and Evans[16] modified for the photoelectric colorimeter by Gibson and Evelyn.[18] In most instances, specimens were obtained from an indwelling arterial needle as described by Noble and Gregersen.[33] This facilitated prompt withdrawal of timed specimens, obviated the danger of stasis, decreased hemolysis, helped preserve veins, and ensured the use of mixed blood. Ampules of the dye solution were obtained from the W. R. Warner Company or made up from dye obtained from the Eastman Kodak Company. Blood volumes were calculated, using centrifuged hematocrits. No correction was made for plasma included in the centrifuged red cell mass.[19] The error of this method is ±175 cubic centimeters.[28] Blood loss at operation was measured by the weighed sponge technique.[44] Accurate records of electrolyte intakes were not maintained, except in those few patients receiving parenteral fluids only for a period before and after operation.

RESULTS

Serum sodium values were below the generally accepted normal values in many of these patients before operation (Fig. 1). The average value for this group of patients was 125 meq. per liter, the range being from 103 to 145 meq. per liter. These findings are in sharp contrast with the serum sodium concentrations found, using the same technique, in fifty normal blood donors whose levels ranged from 132 to 152 meq. per liter, and averaged 139 meq. per liter. The lowest serum sodium determination, 103 meq. per liter, was seen in a debilitated patient who had carcinoma of the esophagus producing total obstruction. Serum potassium determinations before and after operation were within the normal range in all cases except for two patients who showed abnormally low values (Fig. 2). Both of these patients had carcinoma of the esophagus, were severely debilitated, and had received large amounts of intravenous fluids containing inadequate amounts of potassium chloride. Serum chloride concentrations were slightly lower than normal. No significant difference was noted between the pre- and postoperative values (Fig. 3). The average value for the group before operation was 95 meq. per liter. Postoperatively, the level remained about the same, ranging from 110 to 75 meq. per liter.

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*The ossein gelatin used was Knox P 20 type obtained from Knox Gelatine Protein Products, Inc., Camden, N. J.

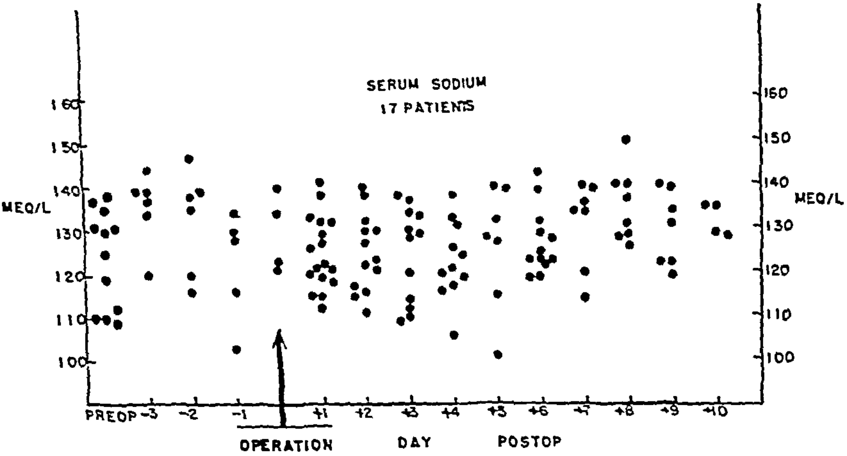

Fig. 1.

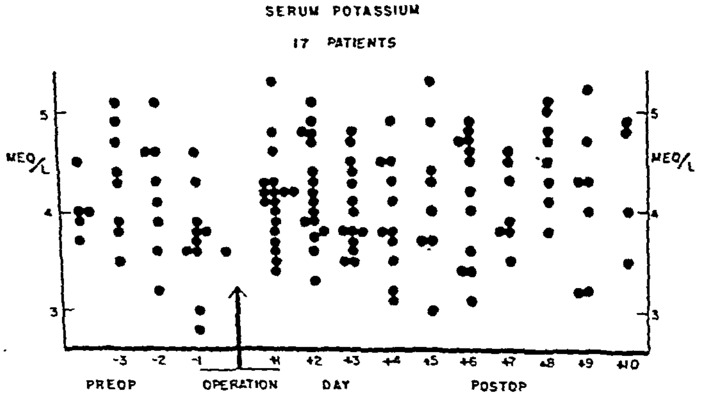

Fig. 2.

Changes in the urinary excretion of sodium were marked and consistent (Fig. 4). Before operation the urinary excretion of sodium ranged from 20 to 240 meq. per twenty-four hours. On the day after operation, the excretion of sodium was always depressed, the range being from 1 to 40 meq. per day. Generally, the depression lasted for about five days, and eventually reached the preoperative level by the seventh or ninth day. In all cases in the immediate postoperative period, the excretion of sodium was less than that of potassium. Urine potassium excretion varied from 10 to 150 meq. per day before operation and there was no significant change in the range of values postoperatively. (Fig. 5). Preoperative chloride excretion ranged from 12 to 230 meq. per day

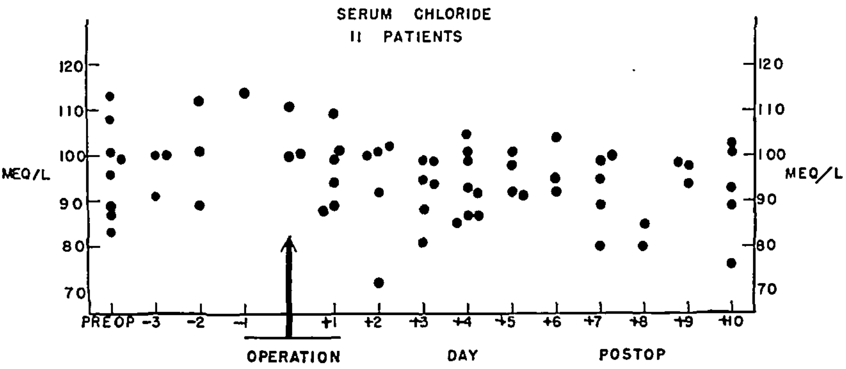

Fig. 3.

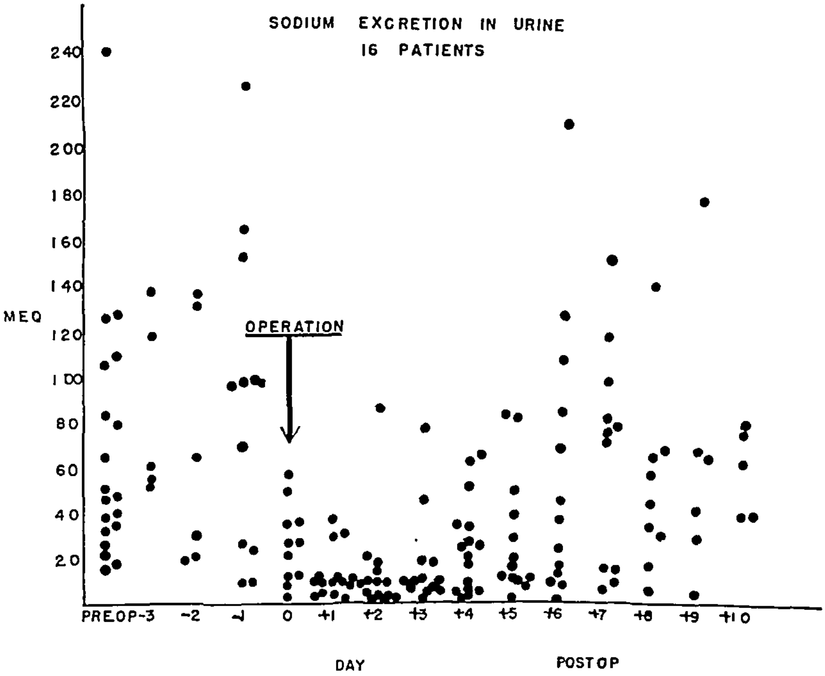

Fig. 4.

(Fig. 6). In most cases, the urinary excretion of chloride was diminished during the first three postoperative days. In two patients chloride excretion was unchanged.

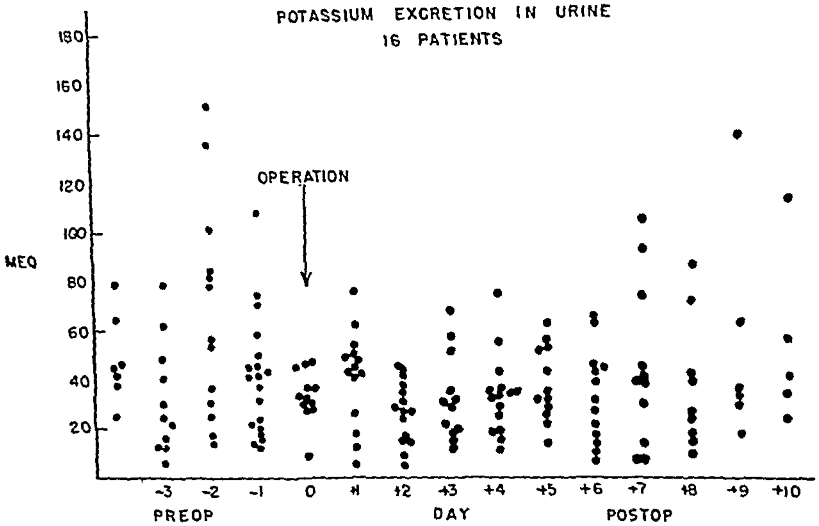

Fig. 5.

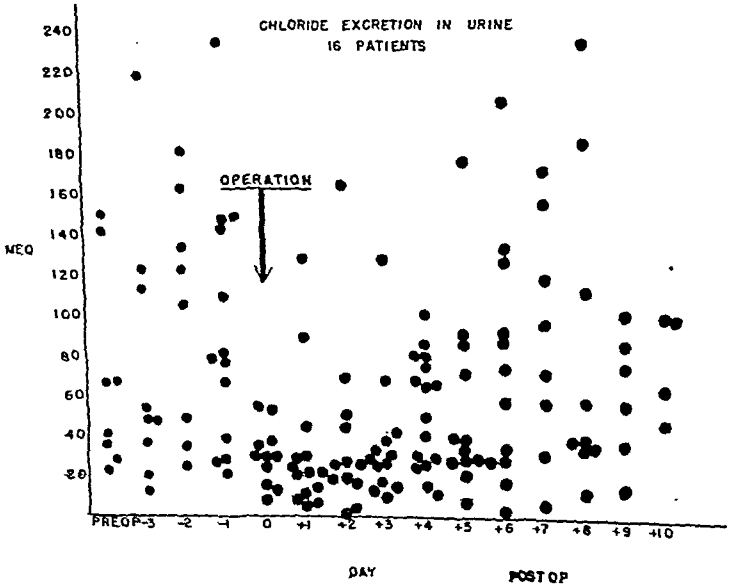

Fig. 6.

In Fig. 7 is shown the average excretion of electrolytes in urine for this group. It can be seen that the sodium excretion was markedly diminished postoperatively; the average figure was 10 meq. during the first twenty-four hours. This depression lasted for five days. The chloride excretion also was diminished postoperatively, but the average chloride excretion was higher than the average sodium value. Postoperative potassium excretion did not differ significantly from the preoperative average.

The average concentration of the electrolytes in the urine followed the same general trend (Fig. 8). Potassium, however, was excreted in greater

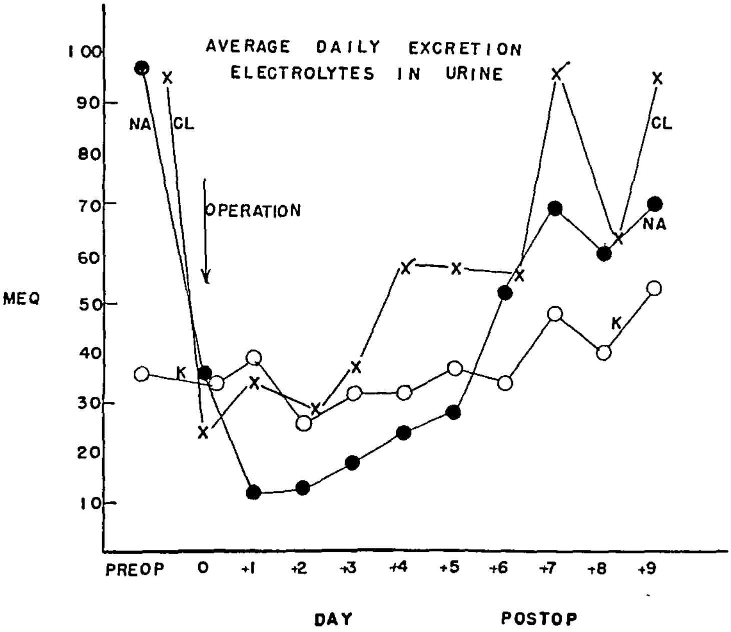

Fig. 7.

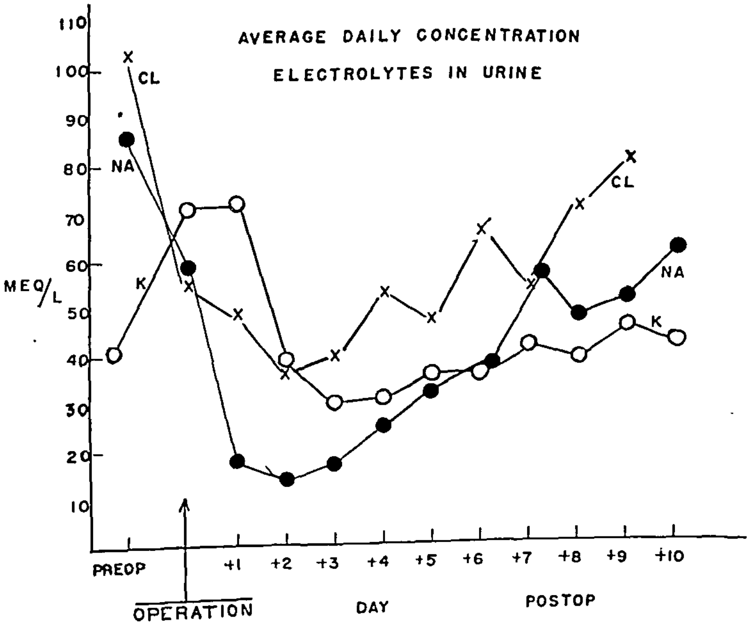

Fig. 8.

concentrations postoperatively than preoperatively. Urine volume decreased slightly postoperatively and therefore the total amount of potassium excreted was about the same before and after operation.

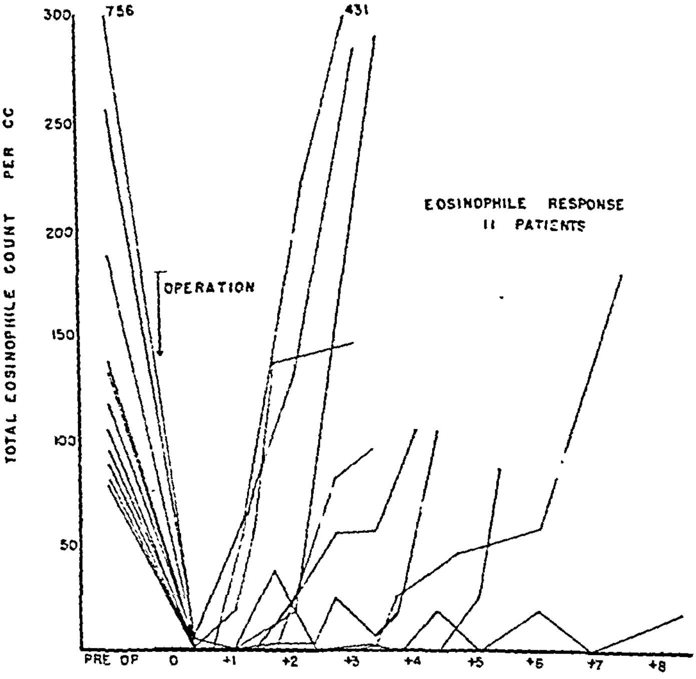

Fig. 9.

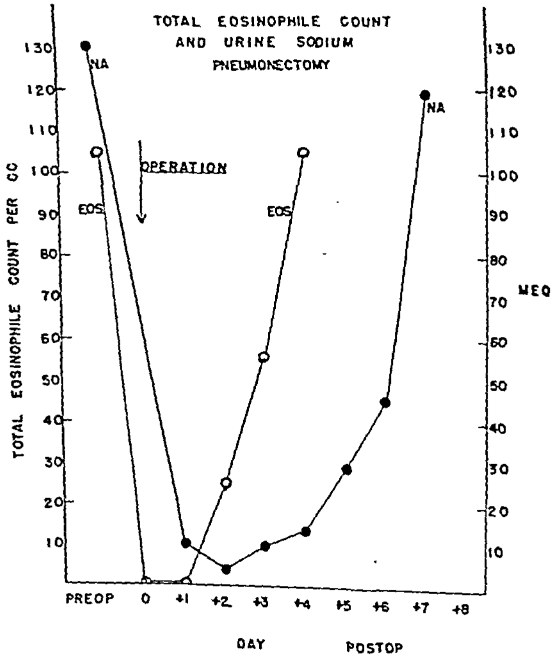

Fig. 10.

Total eosinophil counts were made before and twice daily after operation in 11 patients. The counts fell sharply to six or below after operation in all cases (Fig. 9). A return to normal occurred by the fifth day in all but one patient. This man had a nonresectable esophageal carcinoma and died on the eleventh postoperative day. Depression of the eosinophil count was more prolonged in patients whose clinical condition was poor or who had more severe operations. The response of the eosinophil count to the administration of ACTH was observed in these patients before operation and after the count had returned to normal postoperatively. The response was satisfactory in every case.

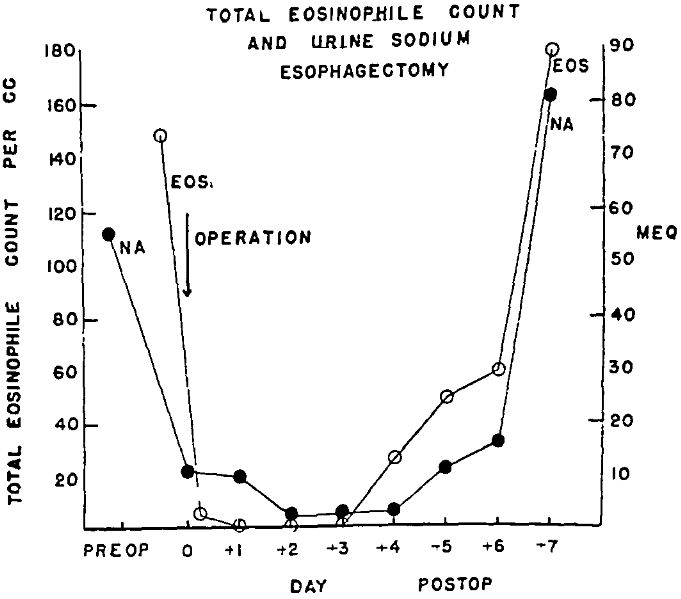

Fig. 11.

In the eleven patients on whom eosinophil counts were done, the return of the eosinophil count and of the sodium excretion to preoperative levels was compared. In six patients the eosinophil count returned to normal much earlier than the sodium excretion. An example of this is seen in Fig. 10. In five patients the eosinophil count followed the sodium excretion. An example of this is illustrated in Fig. 11.

The plasma volume was determined in seventeen patients. It was usually decreased postoperatively in the first eleven patients studied, although blood replacement at operation had been adequate. None of them received plasma or gelatin in the immediate postoperative period. In the last six patients, plasma or gelatin was given to prevent hemoconcentration. The two groups are compared in Fig. 12. Depletion of the plasma volume and hemoconcentration on the first postoperative day can be avoided by the administration of plasma or gelatin during the first twenty-four hours after operation.

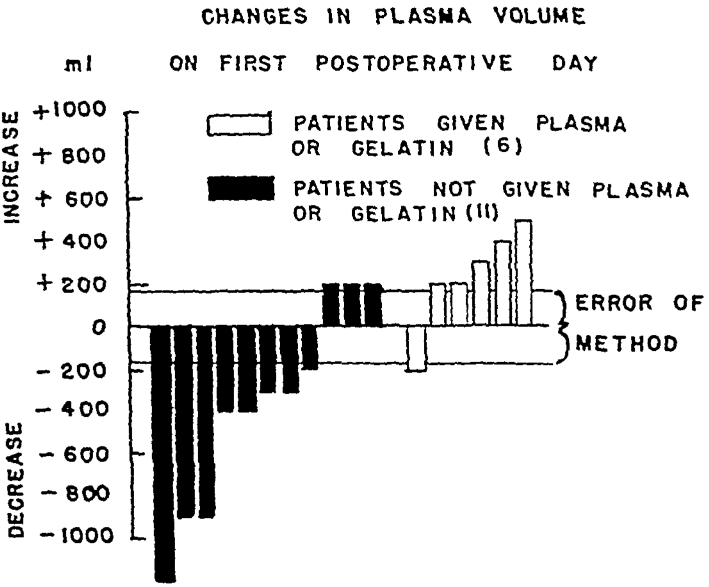
CHANGES IN PLASMA VOLUME
ml ON FIRST POSTOPERATIVE DAY
INCREASE
DECREASE
+1000
+800
+600
+400
+200
0
-200
-400
-600
-800
-1000
PATIENTS GIVEN PLASMA OR GELATIN (6)
PATIENTS NOT GIVEN PLASMA OR GELATIN (11)
ERROR OF METHOD

Fig. 12.

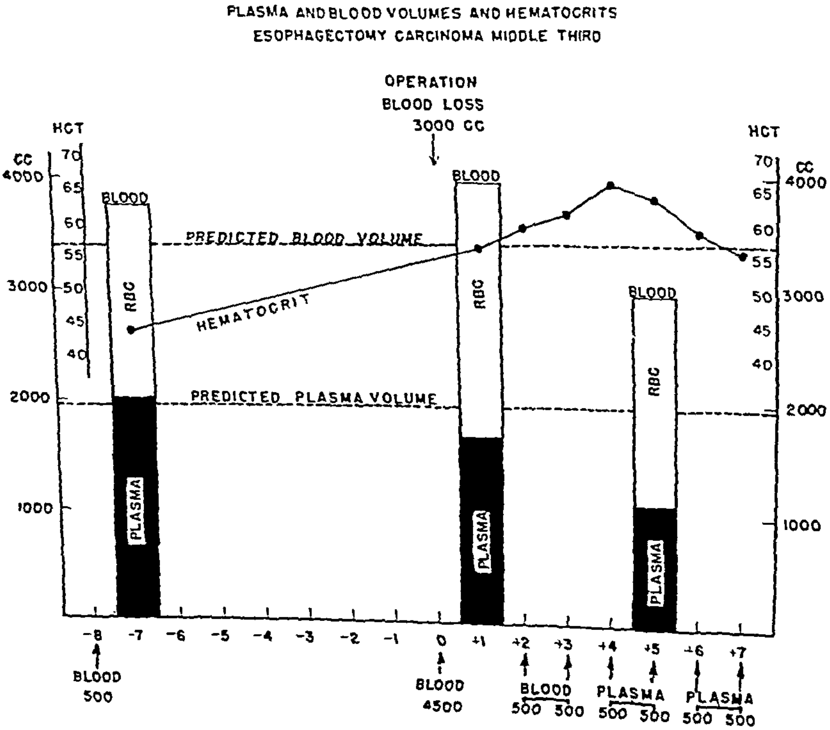
PLASMA AND BLOOD VOLUMES AND HEMATOCRITS
ESOPHAGECTOMY CARCINOMA MIDDLE THIRD
OPERATION
BLOOD LOSS
3000 CC
HCT
CC
4000
3000
2000
1000
70
65
60
55
50
45
40
BLOOD
RBC
PLASMA
PREDICTED BLOOD VOLUME
PREDICTED PLASMA VOLUME
HEMATOCRIT
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
+3
+4
+5
+6
+7
BLOOD 500
BLOOD 4500
BLOOD 500 500
PLASMA 500 500
PLASMA 500 500

Fig. 13.

Three illustrative cases demonstrate this phenomenon. The first case was an esophagectomy (Fig. 13). There was a fall in plasma volume in the postoperative period, with a concomitant rise in hematocrit. This patient was poorly handled in that she received too much blood and no plasma during and immediately after operation. The second patient had a pneumonectomy performed for carcinoma of the lung (Fig. 14). A rise in hematocrit occurred six hours after operation. Following the administration of 1000 ml. of ossein gelatin, the hematocrit fell to normal value. In the third case repeated plasma volume determinations were done (Fig. 15). This patient's initial blood volume was far below the predicted level. He was given 1000 ml. of blood and 300 ml. of plasma before operation. This resulted in a return of his blood and plasma volumes to approximately their predicted normal values which were maintained in the postoperative period.

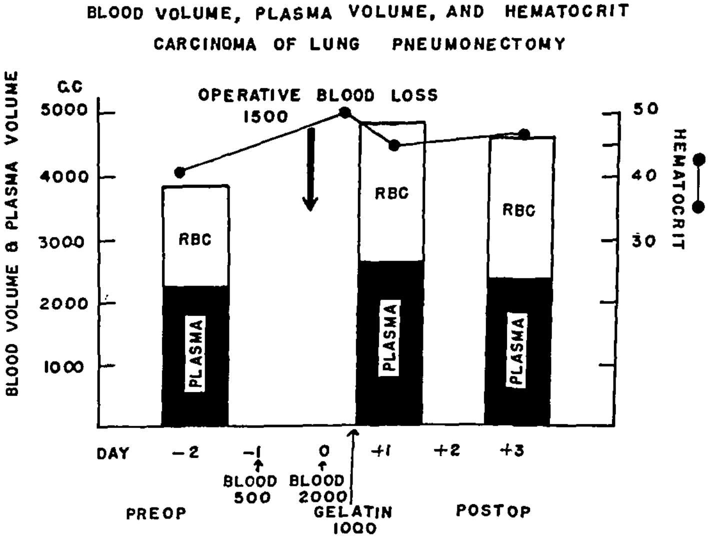

Fig. 14.

DISCUSSION

The low serum sodium levels in this group of patients, both pre- and postoperatively, was an unexpected finding. The cause of the hyponatremia is not clear. Sims and associates[40] have reported asymptomatic hyponatremia in a group of ten patients with chronic pulmonary tuberculosis. In their group of patients, administration of sodium chloride resulted in increased urinary excretion of sodium. On the other hand, when sodium was withheld, urinary excretion diminished. Neither increasing nor decreasing the sodium intake changed the concentration of sodium in the serum. These authors contrasted their findings with those observed in adrenal cortical insufficiency in which urinary sodium excretion continues in the face of low serum levels and low sodium intake. They attributed the findings to a primary intracellular hypo-osmolarity with final adjustment of all electrolyte levels at a lower concentration. That adrenal insufficiency is probably not the cause of the hyponatremia in our patients is indicated by the normal response of the eosinophils to the Thorn test and to the trauma of operation and by the uniform decrease in the urinary excretion of sodium after operation. With one exception, we did not administer large amounts of sodium in the postoperative period to our patients. In one patient who received large quantities of sodium chloride in isotonic solution ofter operation, the urinary excretion of sodium and the serum sodium level remained low (Fig. 16). By the fourth postoperative day, this patient had retained almost 1000 meq. of sodium. Water was retained in proportionate amounts so that approximately one liter was added to the body fluids for each 112 meq. of sodium. This undesirable result of administering large amounts of sodium chloride in the immediate postoperative period has been well shown.[2,7,8,9,31]

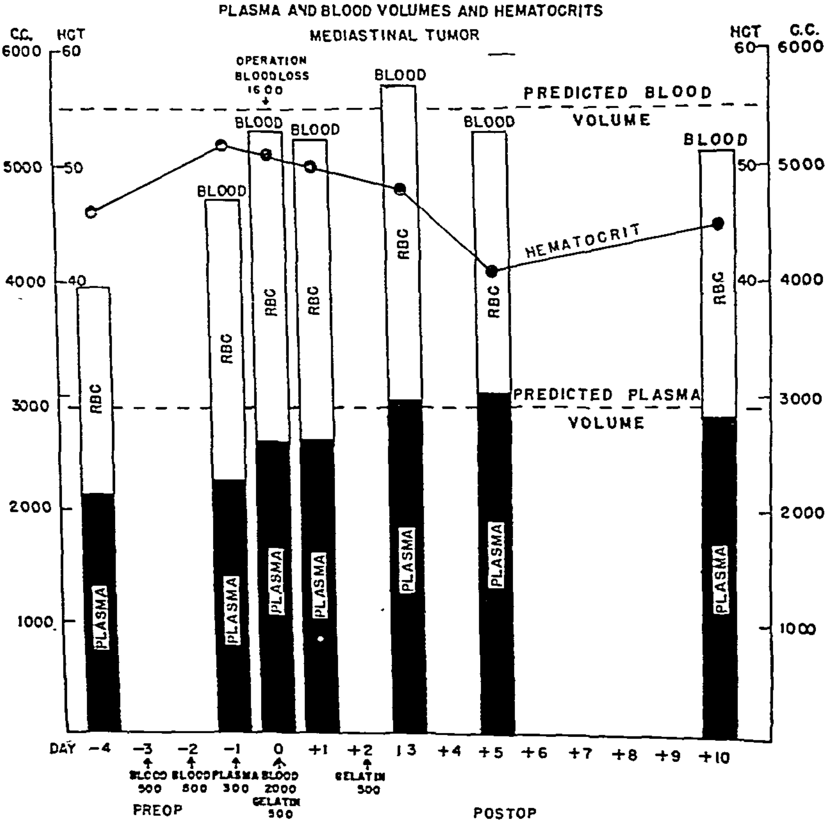

Fig. 15.

Only two patients in this series had low serum potassium levels. Both patients had carcinoma of the esophagus, were severely debilitated, and had received parenteral fluids for long periods of time. They did not exhibit the typical clinical picture of hypopotassemia, nor did they show electrocardiographic changes. The published reports describing potassium deficits in surgical patients concern patients who have lesions of the gastrointestinal tract.[5, 22, 27, 36] Most of the patients in this group were able to take food by mouth early in the postoperative period. Therefore, one would not expect potassium deficiencies to develop.[12] The serum chloride concentrations generally were within the normal range. The patients who had low serum chloride values also had low serum sodium and often low serum potassium values.

The diminished excretion of sodium after operation was striking. The relation of this decreased sodium concentration to increased secretion of the desoxycorticosterone-like hormones of the adrenal cortex has been shown by the studies of Conn, Hardy, Johnson and associates, Leaf and Couter, and Thorn.[10, 11, 20, 23, 25, 41] It has been pointed out that the 11-oxysteroid hormone of the adrenal cortex influences the number of circulating eosinophils.[42]

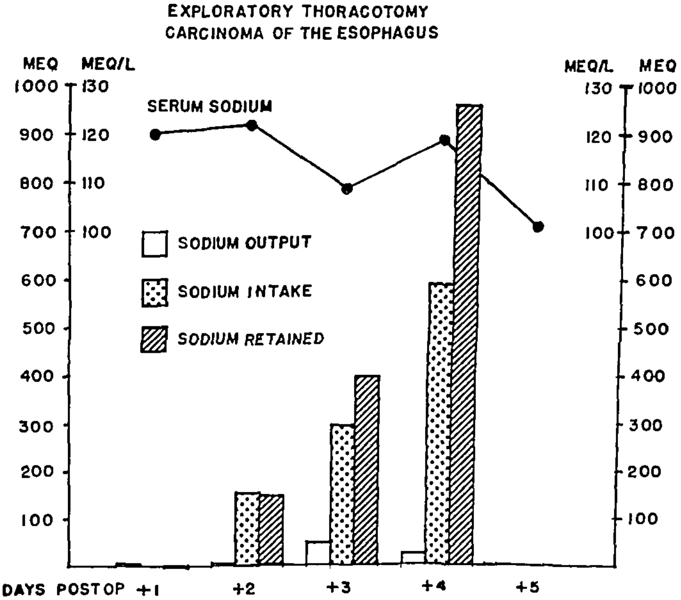

Fig. 16.

Moore[29] has pointed out that these urinary electrolyte changes probably represent a normal protective mechanism. In the majority of the cases reported here, urinary sodium excretion did not reach preoperative levels until the sixth postoperative day. In sharp contrast to sodium excretion the urinary excretion of potassium was not diminished postoperatively. The increased urinary excretion of potassium reported by others was not observed.[5, 22, 27, 36]

Much interest has been shown in the relationship of excretion of sodium and chloride in the urine of surgical patients. VanSlyke and Evans[43] used the Fantus test[14] for urinary chloride concentration as a guide in the administration of sodium chloride, but warned that reliable results could probably not be obtained in the immediate postoperative period. That there is frequently marked difference in the total urinary excretion of sodium and chloride, particularly when acid base balance is disturbed, has been emphasized by Randall and associates.[37] They have shown that dependence upon urinary excretion of chloride alone may lead to serious errors in administration of sodium chloride. In half our patients after operation, there was good correlation between urinary excretion of sodium and chloride. In the remainder, more chloride than sodium was excreted.

The response of the total eosinophil count to the administration of ACTH and the trauma of operation as an indication of the adequacy of function of the adrenal cortex was proposed by Thorn and associates.[42] Hardy[20] showed close correlation between the eosinophil response and the changes in urinary excretion of sodium while Johnson and co-workers[23] have demonstrated the dissociation of these. This dissociation has been emphasized by Moore,[30] and is important because the return to normal of the eosinophil count does not necessarily mean resumption by the kidneys of normal excretion of sodium. This was demonstrated in a number of our patients.

The importance of blood volume determinations in seriously ill surgical patients has been pointed out.[1, 6, 26, 32, 35] In our early cases, blood volumes were determined before and after operation to ensure that preoperative transfusions were adequate and operative blood loss had been properly replaced. A diminution in the plasma volume postoperatively was observed, even though the blood lost at operation had been replaced. The serosanguinous pleural effusion occurring after thoracotomy, as well as the loss of fluid into traumatized areas, is probably sufficient to account for the diminished plasma volume. Plasma or gelatin was given to the later cases after operation to replace this plasma loss and avoid hemoconcentration.

SUMMARY

1. Nineteen patients subjected to major intrathoracic operations were studied. Urine and serum sodium, potassium, and chloride concentrations were determined. Plasma volumes and hematocrits were measured and blood volumes calculated. Total eosinophil counts were made in eleven patients.

2. The preoperative serum sodium concentration was usually low, averaging 125 meq. per liter. Little change was observed after operation. Serum potassium and serum chloride did not show significant deviations.

3. Urine sodium excretion was depressed postoperatively, usually to less than 10 meq. per day. This depression lasted for about five days. Urine chloride excretion also was depressed postoperatively, but not to the same extent. The urinary potassium excretion showed no significant change after operation.

4. There was no evidence of adrenocortical insufficiency in any of these patients.

5. After operation, diminished urinary excretion of sodium persisted longer than depression of the total eosinophil count in half of the patients so studied.

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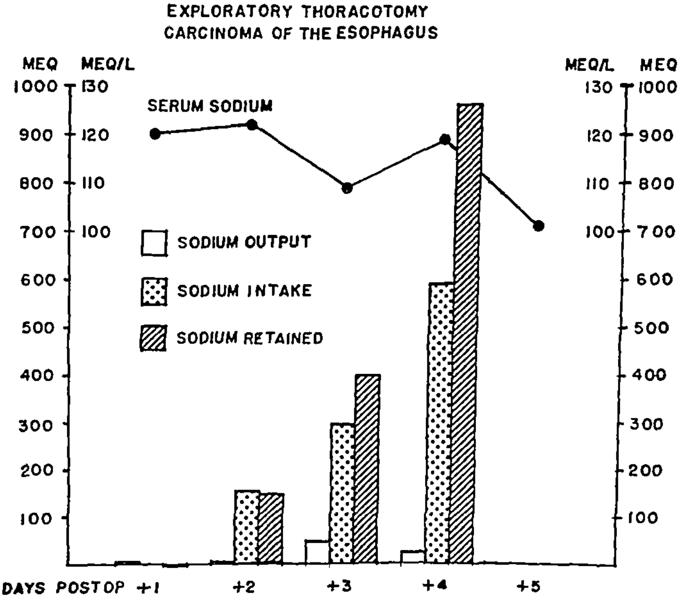

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5. After operation, diminished urinary excretion of sodium persisted longer than depression of the total eosinophil count in half of the patients so studied.

6. Diminution of the plasma volume and increase in the hematocrit was noted during the first twenty-four hours after operation, unless plasma or a plasma substitute (ossein gelatin) was administered.

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DISCUSSION

DR. JOHN H. GIBBON, Jr., Philadelphia.—There are certain results of major operations which follow operation upon the chest, as well as the abdomen or any other part of the body, and one such result is this rather strikingly diminished urinary excretion of sodium postoperatively. We have a series of seventeen cases in which this has occurred in every case. The depression of the urinary excretion of sodium lasted about five days. Most of these seventeen patients were subjected to very prolonged operations. I think it is important to realize that following long operations, the diminished urinary excretion of sodium may last as long as five days. Another finding which we encountered was the low level of serum sodium before operation which was unchanged after operation. This has been previously reported in a small number of cases in patients with chronic tuberculosis. We do not know the explanation for this hyponatremia, but we do know that if you try to raise the low serum sodium level in the immediate postoperative period by administering large quantities of physiologic salt solution, sodium and water accumulate in the body, the urinary excretion of sodium increases only slightly, and the concentration of sodium in the serum is unchanged.

Finally, we have found, as many of you have probably also, that one does not learn a great deal from total eosinophil counts. In general, total eosinophil counts, if they bounce up promptly after a major operation, indicate that the patient is getting along pretty well. If they stay down, it probably is an indication that the patient has received severe trauma and is not in good condition. But, it should be emphasized, the depression of the total eosinophil count does not necessarily parallel the depression of the renal excretion of sodium. It would appear to us that you can regard the eosinophil count as a measurable effect of the 11-oxysteroids of the adrenal cortex, whereas the electrolyte effects are due to the desoxycorticosterone-like hormones, and it is this group which affects the fluid and electrolyte balance postoperatively. You will not necessarily obtain an indication of the activity of the latter hormones from total eosinophil counts.

COMPLICATIONS AND SURGICAL TREATMENT OF HIATUS HERNIA AND SHORT ESOPHAGUS

DONALD B. EFFLER, M.D., AND CHARLES S. BALLINGER, M.D.
(BY INVITATION)
CLEVELAND, OHIO

HIATUS hernia is not thought of as a fatal or even an incapacitating disease. There is a popular concept that this is an innocuous ailment capable only of dyspepsia and other trivial symptoms. Actually this idea is as erroneous as it is widespread. In reality the symptoms of hiatus hernia may be distressing, but the complications of the hernia are serious and occasionally fatal.

Consideration of the complications associated with hiatus hernia is important in recognizing the indications for surgical repair. Esophageal hiatus hernias are observed more commonly with improving diagnostic techniques. Brick[1] has pointed out that the incidence of hiatus hernia among patients undergoing roentgen examination of the upper gastrointestinal tract is approximately 9 per cent. The incidence may prove to be higher in the future. Recognizing the increasing frequency with which true hernias are seen, it becomes more important to develop a sound method of evaluation for therapy. A distinction between the symptoms and complications of hiatus hernia will be helpful in selecting treatment suited for the individual case.

Hiatus hernia is commonly classified into three main groups:[2]

1. The paraesophageal hernia.
2. The pulsion type hernia.
3. The short esophagus with thoracic stomach.

We believe that a sharp distinction must be made between true hernias of the paraesophageal or pulsion type and the thoracic stomach associated with short esophagus. This distinction is based on our belief that the etiology differs between the two groups as do the related symptoms, complications, and clinical course. For clarity the two groups will be presented separately.

COMPLICATIONS OF HIATUS HERNIA

Whereas the symptoms of true hiatus hernia are extremely variable in character and degree, the complications are rather clearly defined; for the most part we can classify them under three major headings: (1) bleeding; (a) occult or massive, (b) hematemesis or melena; (2) peptic ulcer; (3) volvulus.

Bleeding: In all probability this is the most common complication of hiatus hernia. Much has been written on this subject, calling attention to the

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From the Cleveland Clinic and the Frank E. Bunts Educational Institute.

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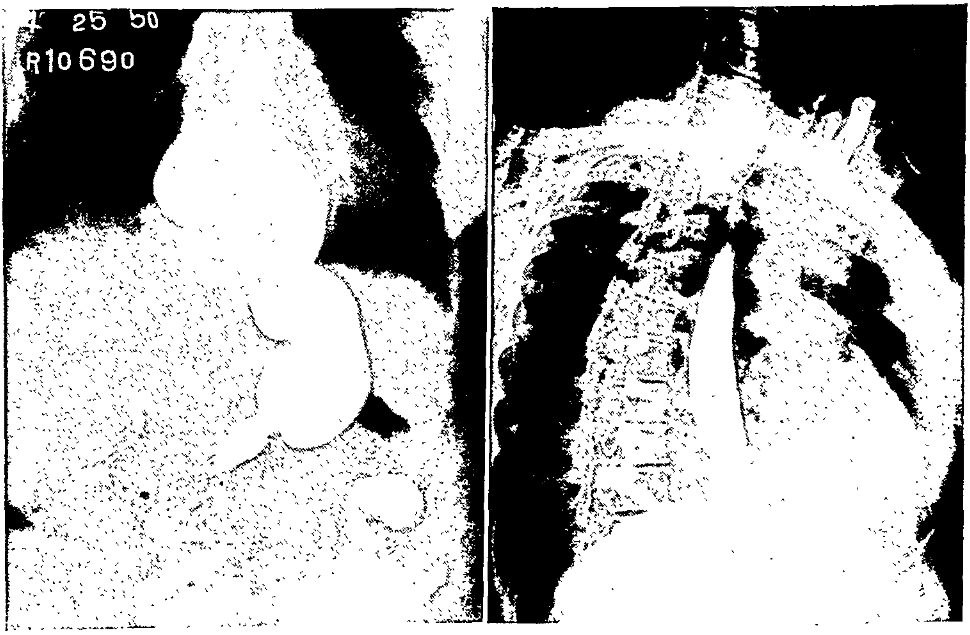
"blood loss" anemia, fatal hemorrhage, intermittent hematemesis, and melena attributable to hiatus hernias.[3-7] Theories as to the cause of the bleeding have included erosion of gastric mucosa associated with venous congestion, gastric varices, peptic ulcer, esophagitis, and intestinal defects secondary to the impaired gastric physiology.[7, 8] Regardless of the causes, bleeding is a frequent and troublesome complication of hiatus hernia. It may be occult in character and manifested only by "blood loss" anemia; on the other hand, there may be massive bleeding without premonitory signs. Hemoglobin determinations of less than 6.5 Gm. are not uncommon among this group of patients. It may be pertinent that hemorrhage does not seem related to the size or shape of the hernia.[6]



A. *B.*

Fig. 1.—*A*, Hiatus hernia associated with volvulus manifested by upper gastrointestinal obstruction. The patient was a 61-year-old woman with progressive increase of symptoms over a ten-year period. *B*, Postoperative barium study one year after operation; complete relief of all symptoms.

Peptic Ulcer: True peptic ulcer has been recognized for many years as a complication of hiatus hernia.[9] The cause of the ulcer has been attributed to erosion, trauma, retained acid secretions, and other factors. Ulcers may

TABLE I*

	NO. OF PATIENTS
Hematemesis	7
Melena	9
Blood loss anemia (below 11 Gm.)	14
Critical blood loss (below 7 Gm.)	5

*Of the 36 patients operated upon for hiatus hernia, 14 manifested complications by bleeding. Accurate evaluation is difficult as all patients received supportive therapy before transfer to the Cleveland Clinic.

be difficult to demonstrate by barium studies; likewise, their response to medical therapy is difficult to evaluate. Bleeding from these ulcers is common; perforation is not, although it usually has been reported as fatal.[10]

Volvulus: This term is broadly used to include twisting or folding of herniated stomach within the mediastinum. For simplicity we have included incarceration with obstruction under this heading, although true volvulus need not be a factor. Usually this complication is manifested on the roentgenogram by two or more fluid levels in the herniated segment.[11] Occasionally it is demonstrated by herniation of the antrum with the fundus and pylorus seen below the hiatus. Clinically these patients are more inclined to vomiting, pain, and dysphagia.[12-14] We have not seen strangulation of the incarcerated segment, although it has been reported.[11]

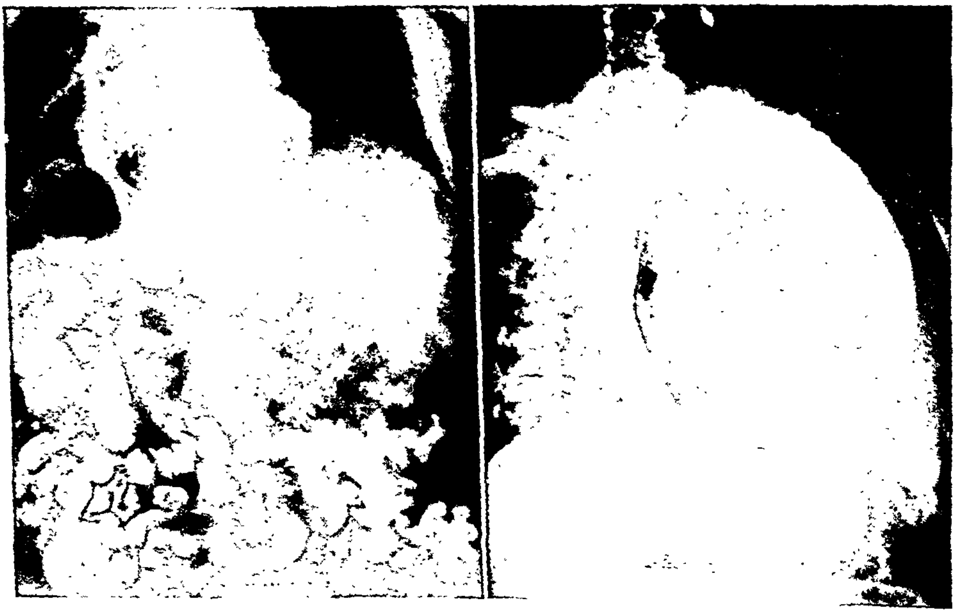

A. *B.*

Fig. 2.—*A*, Large paraesophageal hiatus hernia complicated by volvulus and severe bleeding; symptoms present twenty-one years. On admission to hospital hemoglobin was 6.2 Gm. *B*, Postoperative roentgenogram eight months after surgery; complete relief of symptoms. Hemoglobin 13.4 Gm.

Comment: By no means are all complications of hiatus hernia included in this classification. The overwhelming majority, however, will fall into one or more of the three categories.

Many of the voluminous reports found in the literature discuss briefly the subject of treatment or indications for either medical or surgical approach. Frequently encountered is the suggestion of correlation between the size of the hernia and severity of symptoms. We do not believe that such relationship exists; nor do we feel that size alone is an indication of the type of treatment. A small hernia may be reponsible for an exsanguinating hemorrhage, whereas a much larger hernia may produce nothing more than occasional eructation.

Specific indications for surgical repair of hiatus hernia will always be variable. Careful evaluation of the patient, however, with distinction between symptoms and complications, will assist in election of either medical or surgical therapy. We believe that the presence of one or more of the complications listed is an indication for surgical repair.

COMPLICATIONS OF SHORT ESOPHAGUS

The symptom complex produced by short esophagus with thoracic stomach may parallel that of hiatus hernia. The problem of differentiation between the two lesions is well known. As might be expected, there is a similarity in the range of complication.

Again we have stressed three separate categories of complications attributable to short esophagus. These are: (1) stricture; (2) bleeding: (a) occult or massive, (b) hematemesis or melena; (3) peptic ulcer: (a) gastric or esophageal, (b) penetrating or perforating.

Stricture: Dysphagia is the most common symptom associated with short esophagus, and in itself is not a complication. In advanced cases, particularly those associated with esophagitis or peptic ulcer producing cicatricial contracture, a fibrotic stricture may be produced that resists all efforts by bouginage and medical therapy.[15, 16] Severe pain and chronic inanition, secondary to the stricture, may render the patient a gastrointestinal cripple. In such cases differential diagnosis between carcinoma and inflammatory stricture may be impossible.

Bleeding: This is a common manifestation and may occur in the variety of ways demonstrated in hiatus hernia.[15, 17] In short esophagus, however, there is an additional bleeding source found in the terminal esophagus. The severe inflammatory changes produced by long-standing esophagitis greatly increase the vascularity of the involved segment. The diseased mucosa is friable and hyperemic, bleeding readily with minimal trauma. Chronic "blood loss" anemia is a common finding in these patients; recurrent episodes of hematemesis and melena are not infrequent.

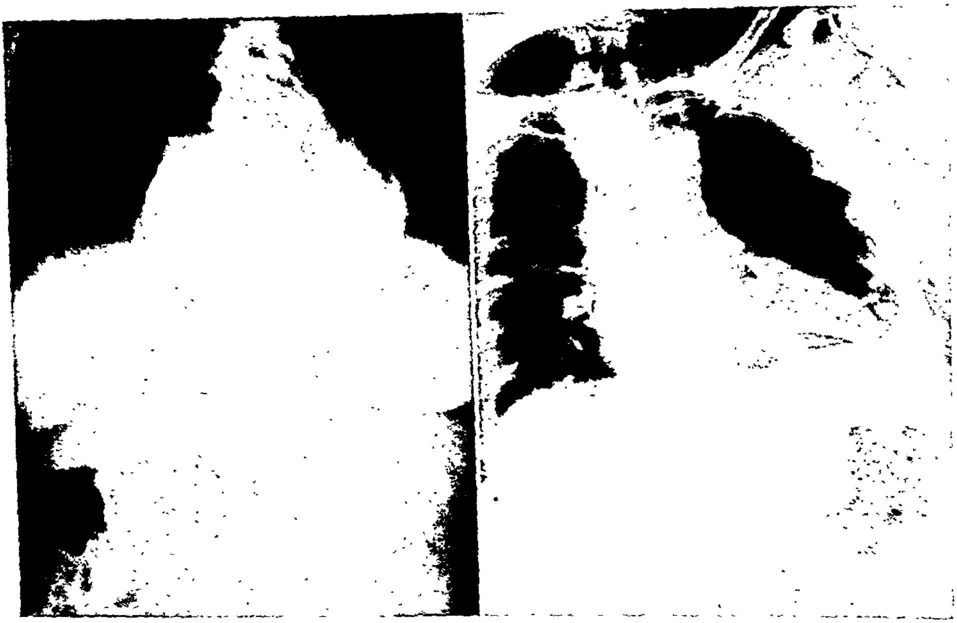
TABLE II*

	NO. OF PATIENTS
Blood loss anemia (below 11 Gm.)	7
Critical blood loss (below 7 Gm.)	4

*Seven of the 16 patients operated upon for complicated short esophagus had episodes of bleeding.

Peptic Ulcer: The association of ulcer with acquired short esophagus has been known for many years. Johnstone, Allison, Dick, and others[18-20] have commented on the severity of the symptoms and the resistance to conservative therapy.

Peptic ulcer may occur as either the initiating factor or as the secondary complication of short esophagus. With severe gastritis and esophagitis, accurate localization by radiographic and endoscopic means is difficult. Although these ulcers have been considered esophageal in origin, findings at operation suggest that they arise more commonly in the cardia of the thoracic stomach. In three of our cases the ulcers had perforated into the mediastinum: one sealed perforation involved the pericardium; another had penetrated the medial segment of the right lower lobe.



A. B.

Fig. 3.—*A*, Large paraesophageal hiatus hernia with extreme volvulus and anterior displacement of esophagus. The patient was a 66-year-old white woman who complained of pain and vomiting associated with ingestion of food. Patient was addicted to paregoric. *B*, Postoperative appearance fourteen months after surgery; complete relief of all symptoms.

Comment: Symptomatic short esophagus is a structure deformity. In the complicated cases conservative treatment has, in many instances, proved inadequate. It may be also impossible to exclude carcinoma by radiographic studies or esophagoscopy. The generally unsatisfactory response to conservative measures, plus the uncertainty of diagnosis, have prompted more radical approaches in selected cases. Consideration of the factors complicating this disease will aid in the selection of candidates for surgery.

SURGICAL TREATMENT

Hiatus Hernia: Thirty-six patients with paraesophageal or pulsion type hernia have been operated upon; recovery has been complete and uneventful in all cases. Each patient manifested one or more complications of hiatus hernia, the presence of the complication having been considered an indication for surgery. Although sufficient time has not elapsed to permit accurate evaluation of indications or techniques employed, the immediate results are uniformly good. All patients have been relieved of symptoms attributed to the hernia, and there have been no recurrences.

A simple technique of surgical repair has been used in these cases, employing the transthoracic approach. In many respects our concept of the hernia and its repair is in accord with the ideas expressed by Allison.[22] However, we have attached greater significance to permanent reduction of the stomach below the diaphragm and less to the so-called sphincter action of the hiatus. A brief description of the operative technique follows:

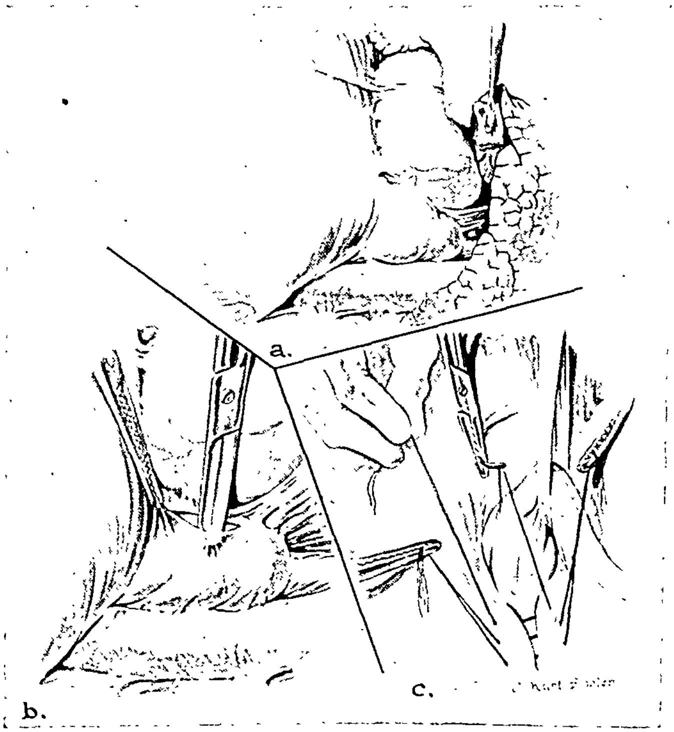


Fig. 4.—*A*, Artist's description of operative field through thoracic approach. *B*, Beginning of mediastinal dissection with incision of mediastinal pleura. Mobilization of esophagus and thoracic stomach is best accomplished by sharp dissection. *C*, Interrupted sutures to approximate the right and left crura are carefully placed after mobilization and dissection are completed.

Endotracheal anesthesia is employed; the patient is placed in a lateral position. The thoracic incision is made over the eighth rib. The mediastinal pleura is incised from the hiatus through the pulmonary ligament to the inferior pulmonary vein. By sharp and blunt dissection the herniated segment and the terminal esophagus are mobilized. The fascia and reflected peritoneum are transected over the hernia and the crura freed by sharp dissection. The retroperitoneal portion of the stomach is gently dissected away from the posterior aspect of the hiatus. After carefully placing the sutures, the right and left crura are approximated posteriorly to the esophagus. The hiatus is closed snugly around the esophagus. A 16 French stomach tube is left in place for twenty-four hours. The phrenic nerve is not disturbed.

Short Esophagus: The need for a definitive surgical procedure in the treatment for short esophagus is evident. Ideally, this procedure should be simple and adaptable to all variations of the disease. The pathologic variations produced by short esophagus, however, have forced us to subdivide the entity and its therapy into two groups: (A) ulcerated short esophagus, (B) simple short esophagus.

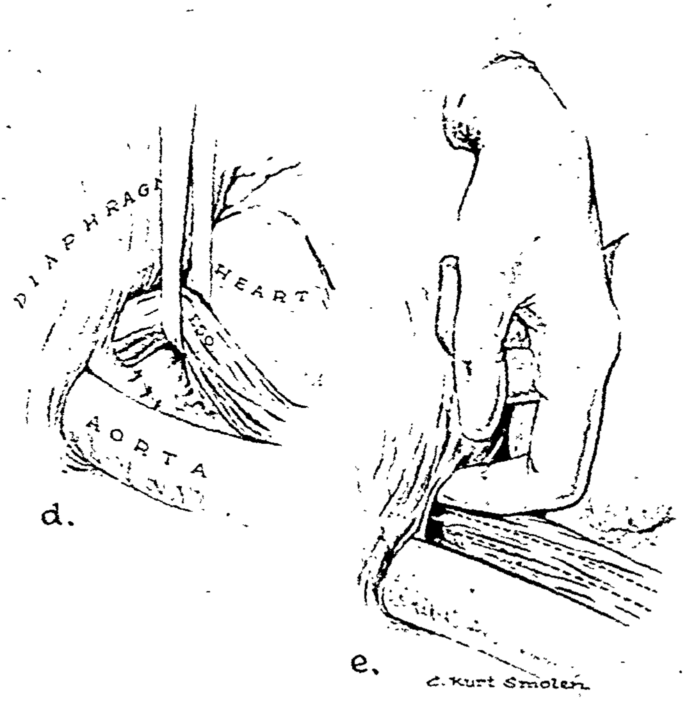


Fig. 5.—*D.* Appearance of operative field at completion of operation. The diaphragm is not paralyzed. *E.* A gastric tube is left in place for twenty-four hours. The operator's index finger examines the hiatus to assure complete closure.

Group A is made up of the cases with extensive shortening, severe periesophagitis, peptic ulcer with penetration, and secondary changes in the surrounding mediastinum. Radiographic studies in this group resemble those of extensive malignancy, and differential diagnosis may be exceedingly difficult. These patients respond poorly, if at all, to conservative measures.

Surgical therapy in this group is a difficult and occasionally hazardous undertaking.[16] The mediastinum surrounding the diseased area is altered by the chronic inflammation. Increased vascularity and fibrosis render the dis-

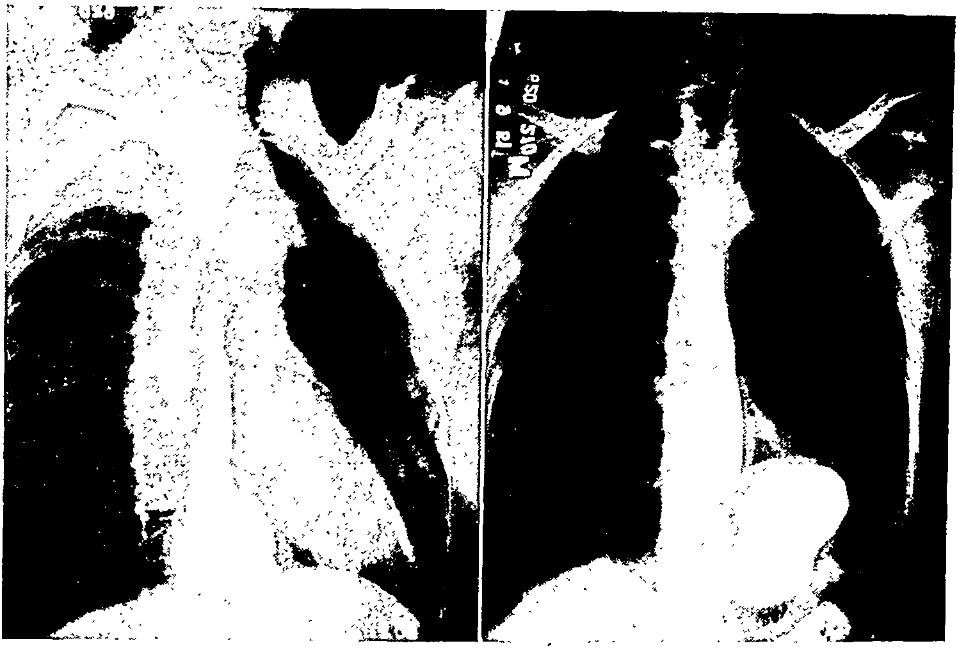


Fig. 6.—*A*, Short esophagus and thoracic stomach in a 60-year-old white woman manifesting severe pain and intermittent obstruction. *B*, Roentgenogram made two months after surgery, illustrating the reduction of stomach within the abdominal cavity. Complete relief of symptoms.

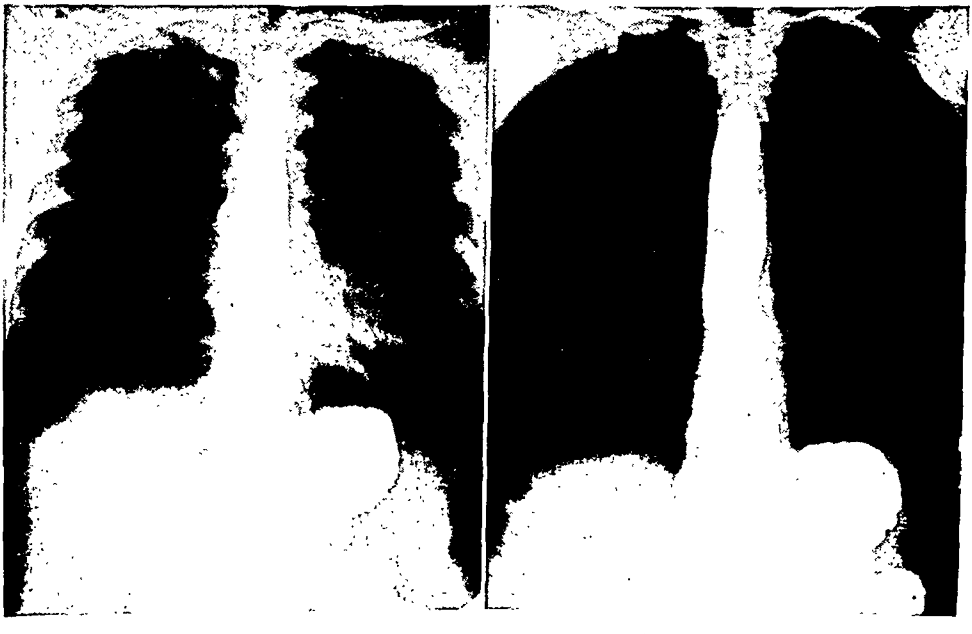


Fig. 7.—*A*, Short esophagus complicated by gastric ulcer in a 55-year-old white woman. Immediate relief following surgical correction without resection. *B*, Short esophagus with thoracic stomach complicated by a large gastric ulcer in a 55-year-old white man believed to have acquired esophageal shortening.

section most tedious. In essence, the surgical treatment of the severely ulcerated short esophagus is similar to that of carcinoma. The diseased segment is resected and continuity is restored by esophagogastrostomy. We have employed this procedure in only six cases: all of the patients were benefited.

The second group includes patients having a short esophagus who have not suffered the severe structural changes which characterize Group A. Whereas one or more of the complications previously discussed may be present, the patient is not reduced to the status of an invalid. Nevertheless, these people require constant therapy and clinical observation as long as the short esophagus remains. The usual medical therapy abetted by bouginage may alleviate but not cure the disease.

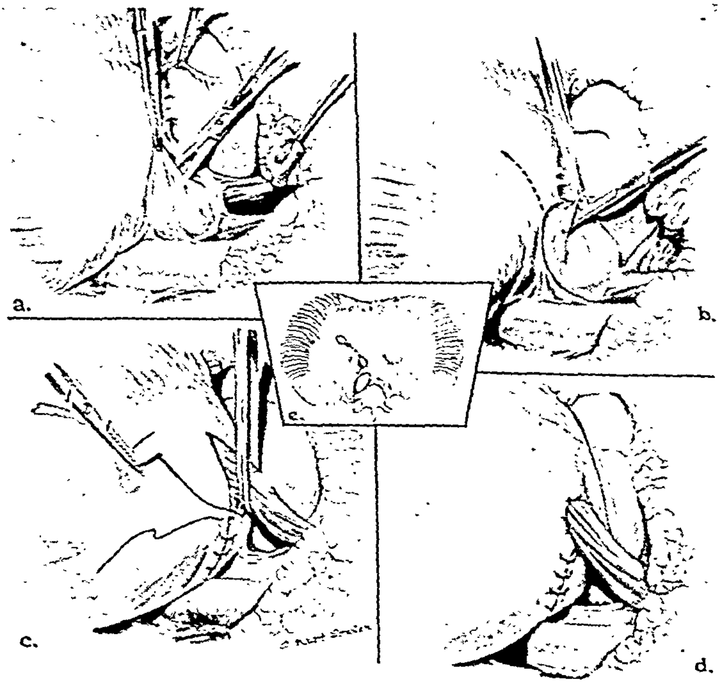


Fig. 8.—Technique of thoracic correction of simple short esophagus. *A*, Interruption of the phrenic nerve and mobilization of thoracic stomach with terminal esophagus by sharp dissection. *B*, Incision of phrenoesophageal fascia and reflected peritoneum over the thoracic stomach. The dotted line illustrates the radial incision through the paralyzed diaphragm. *C*, Posterior approximation of crura following reduction of thoracic stomach. *D*, A completed operation illustrating displacement of esophagus to new hiatal opening at high portion of paralyzed diaphragm. *E*, Diagram illustrating transplanted esophageal hiatus.

Surgical measures for short esophagus have been rather limited. Phrenic nerve crush, gastrostomy, vagotomy, enlargement of the esophageal hiatus, and other procedures have been used with indifferent results. It is not illogical to presume that relief of symptoms and prevention of future complications would depend upon correction of the primary structural abnormality. In other words, unless the stomach has been permanently reduced below the diaphragm and the esophagus of sufficient length to span the vertical diameter of the thorax, the underlying problem remains the same. A *simple operation* has been employed for the past two years in a group of ten patients with short esophagus. The technique is similar to that described by Merendino, Varco, and Wangensteen[21] in the treatment of hiatus hernia. The results have been uniformly good; there have been no failures and no surgical complications. Description of the technique follows:

A thoracotomy incision is made over the eighth rib. The mediastinal pleura is incised; the thoracic stomach and terminal esophagus are carefully mobilized. The phrenic nerve is divided for permanent paralysis of the diaphragm. At this point the surgeon will observe noticeable elevation of the tendinous dome; the hiatus is not appreciably altered in size or position. The site of the "new" hiatus is selected at the dome of the diaphragm and a radical incision made to the esophagus. If mobilization is adequate, the tendinous leaves can be elevated to the cardiac esophagus, reducing the thoracic stomach to the abdominal cavity. The "old" hiatus is closed by approximating the crura. The terminal esophagus is carefully tacked with silk mattress sutures to the tendinous diaphragm to prevent sliding. With this procedure the esophagus is not elongated to a significant degree; in reality the abdominal cavity is lengthened and thoracic space is shortened. In reducing the distance traversed by the thoracic esophagus a former short esophagus may now be adequate in length.

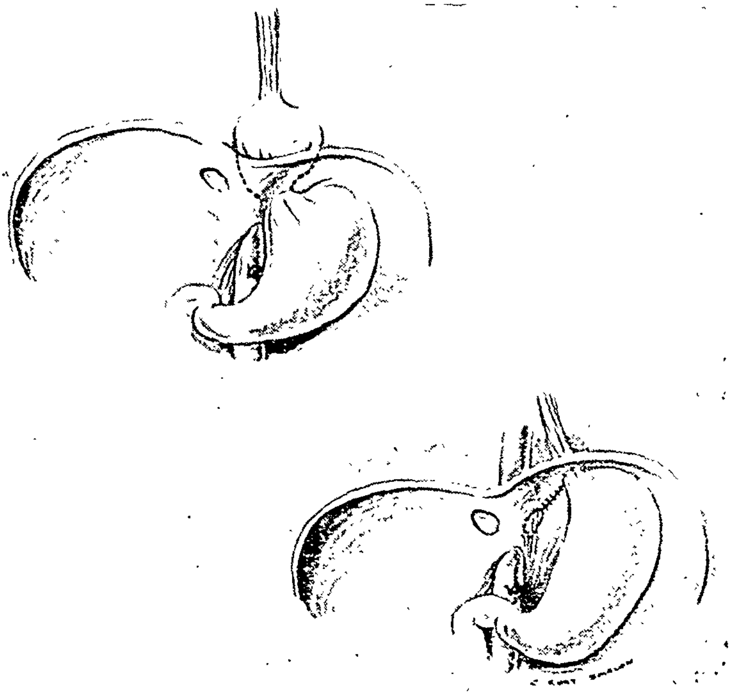


Fig. 9.—Schematic correction of simple short esophagus by permanent paralysis of the left diaphragm and transplantation of esophagus to new hiatus. The short esophagus is not appreciably lengthened, although the thoracic stomach is reduced below the diaphragm.

CONCLUSION

Hiatus hernia may be a serious, incapacitating disease. Surgical correction is not difficult and offers possibility of cure.

Short esophagus is a disease characterized by annoying symptoms and capable of serious complications. Unless the structural defect is corrected permanently, any other form of therapy must be considered as merely palliative. Two methods of surgical treatment are suggested.

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DISCUSSION

DR. FORRESTER RAINE, Milwaukee, Wis.—I began, some five years ago at the Veterans Hospital at Wood, Wisconsin, seeing every patient with a diagnosis of short esophagus and hiatus hernia. I had planned to write a paper on the subject, but I found each year that I knew less than I did the year before and, as a result, have not gotten to it yet.

In all, we have seen eighty-nine patients with a short esophagus and hiatus hernia. Of this group, 50 per cent were so nearly symptomless that no treatment was considered. In the remainder we have the various types of complications that Dr. Effler has talked about. Some of them have a relatively minor stricture, and those we have been able to manage only by dilatation and medical ulcer management. Others have had so much stricture that it could not be overcome by dilatations; they had peptic ulcers at the junction (mos tof those I have seen have been at the junction of the esophageal gastric mucosa rather than in the gastric pouch itself) and some type of resection had to be done. I have also found that there is a very high percentage of associated duodenal or gastric ulcer with the short esophagus cases that have an ulcer and a stricture, and these we have elected to handle by doing a gastric resection with repair of the hernia. I am sorry to say that in my hands none of these procedures have been entirely successful. We have had esophageal ulceration or esophagitis continue after gastric resection, so that we have had a very difficult time feeding them.

The associated vagotomy with resection I feel is some protection against recurrent ulceration at the anastomosis, and that has not as yet occurred in any of our cases. I thought that it might be interesting to show a patient who, I think, demonstrates very well the terrific pull of the inflamed esophagus and how it can shorten and pull a narrow tube of stomach up into the chest.

(Slide.) Here you see a patient who came in with a massive hemorrhage, a duration of four or five years' difficulty in swallowing, and I think you can see the height of the stricture and the ulceration present at the point of stricture.

(Slide.) This shows the patient after he had been treated by dilatation and ulcer diet, and there is much less spasm, and the narrowed area has come down far closer to the diaphragm. Repeated dilatations, however, failed to control him, and finally resection was done.

(Slide.) I think you will also notice the difficulty I have had with him afterward, that is, the amount of gastric retention which is associated with vagotomy. This has been a troublesome feature in some of these resections.

(Slide.) This is a somewhat different type which I think shows some of the difficulties in differential diagnosis between achalasia, carcinoma, and a short esophagus. This is a picture of a short esophagus with ulceration at the junction, rather than early achalasia or carcinoma.

DR. ALTON OCHSNER, New Orleans.—There is no question that many of these patients will require resection. It is of interest to me that when one resects for short esophagus, one leaves an esophagus which is even shorter than before, yet with very few exceptions the results are good after the resection.

Several years ago before this Association I mentioned a patient who had a short esophagus with ulceration, in whom we advised a resection. This patient, an elderly woman, refused resection and, on the advice of one of the younger house officers, we did a phrenicectomy. The patient was promptly relieved of her symptoms and has remained well. Since that time we have done an increasing number of phrenicectomies in these individuals. I am inclined to believe that unless there is considerable damage, the symptoms can be relieved by phrenicectomy, and probably the symptoms in the short esophagus, when they have symptoms, are due to the pull of the diaphragm upon the segment of stomach which is herniated into the thoracic cage.

DR. LAMAR SOUTTER, Boston.—I think that Dr. Effler's paper was very good, particularly in demonstrating variations in technique to suit the length of the esophagus. Several surgeons at the Massachusetts General Hospital have employed a somewhat similar technique with good results. In some ten cases, we have deliberately made an opening in the tendinous portion of the diaphragm sufficiently large to permit the hand to enter the abdominal cavity. This permits exploration of the upper abdomen in an effort to determine the presence or absence of peptic ulcers and gall bladder disease. It is helpful in ascertaining that the stomach has been completely reduced. It is most valuable in permitting accurate placement of the sutures in the diaphragm by bimanual palpation. For those patients with poor crurae posteriorly or with a true short esophagus, the incision can be extended and the esophagus transplanted anteriorly in the manner which Dr. Effler has shown. We have felt that this approach has made the surgery of diaphragmatic hernias easier and more effective. It has not compromised the diaphragmatic muscle nor increased the postoperative morbidity.

DR. EFFLER (closing).—I would like to thank Dr. Raine, Dr. Ochsner, and Dr. Soutter for their kindness in discussing this paper. In reference to the ulcerative or complicated form of short esophagus, I must admit that my own experience is brief, encompassing a period of only two and one-half years. Six of these patients have been treated by resection and esophagogastrostomy. I agree with Dr. Raine and Dr. Ochsner that this is substitution of one disease for another. Dr. Ochsner has suggested that a phrenic crush might be of value in such a patient when pain is the predominant factor. Three of the six patients mentioned above had previous phrenic operations and had transient relief or none at all. It is also worthy of mention that two of the six patients were addicted to codeine or morphine, and two others were in the habit of using large amounts of paregoric. Perhaps the most important argument in favor of primary resection is the difficulty of differentiation between peptic ulcer and primary carcinoma.

As Dr. Raine mentioned, the secondary vagotonic stomach may be a serious problem in patients treated by primary resection. Two of our patients required further surgical assistance following esophagogastrostomy.

Whereas our experience is limited, we are, nevertheless, very optimistic that correction of the simple type of short esophagus with thoracic stomach may prevent the development of future complications associated with severe esophagitis, mediastinitis, and ulceration. We believe that this simple operative correction may well prevent the necessity of primary resection and esophagogastrostomy at a later date.

FORMATION OF A TEMPORARY EXTERNAL ESOPHAGEAL FISTULA OVER A T-TUBE FOR STENOSING ESOPHAGITIS

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CHICAGO, ILL.

STENOSING esophagitis with or without perforation is not a rare condition. Although the etiology is uncertain, the most probable cause is the effect of gastric juice upon the esophageal wall. Whether this gastric juice is a reflux into the esophagus from the stomach or whether it is produced in the esophagus itself because of heterotopic gastric mucosa is still controversial.[1]

The diagnosis of esophagitis should not be overlooked since the symptoms *are so commonplace. Winkelstein,[2] in 1935, reported on a group of cases* which he described as peptic esophagitis; Vinson and Butt,[3] Olsen,[4] and Allison,[5] have attested to its frequency. "Heartburn," a frequent complaint, is described by the patient as a burning sensation in the epigastrium or lower sternal region. Marked substernal pain referred to the throat, the neck, and the arms is seen in the more severe and far-advanced case. Moersch and Camp[6] are of the opinion that this type of pain is due to a diffuse esophageal spasm.

Many cases of mild esophagitis will subside and leave no sequelae, however, should the condition progress and become chronic, cicatrization of the lower part of the esophagus occurs. Olsen and Harrington[7] believe that such cicatrization may lead to actual shortening of the esophagus. It is questionable whether or not hiatal hernia should be considered a consequence of rather than the cause of esophagitis.

Of interest is the fact that these cases are frequently associated with gastroduodenal ulcers. Van Aken[8] reported three cases of stenosing peptic esophagitis, all of which had previous gastric resections for duodenal ulcers. He is of the opinion that the treatment of choice should be resection of the involved part of the esophagus.

Conservative treatment should be attempted in all cases of esophagitis prior to surgery. Such therapy consists of the neutralization of gastric secretions, elevating the patient's head, antispasmodics, and dietary regimes. If a cicatricial stricture occurs, dysphagia may be relieved by cautious dilatation in the hands of an expert, and should this fail resection may be indicated.

Wangensteen and Logan Leven[9] reported on six cases of esophagitis; they recommend subtotal gastrectomy as rational therapy to reduce the secreting surface of the stomach.

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Read at the Thirty-first Annual Meeting of the American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

When the pathologic process has extended to such a point as to make resection impossible (chronic perforation with spillage, extensive fibrosis, and edema), the only recourse might be the establishment of an external esophageal fistula over a T-tube. After a perusal of the literature, no report could be found wherein a similar procedure was done for stenosing esophagitis.

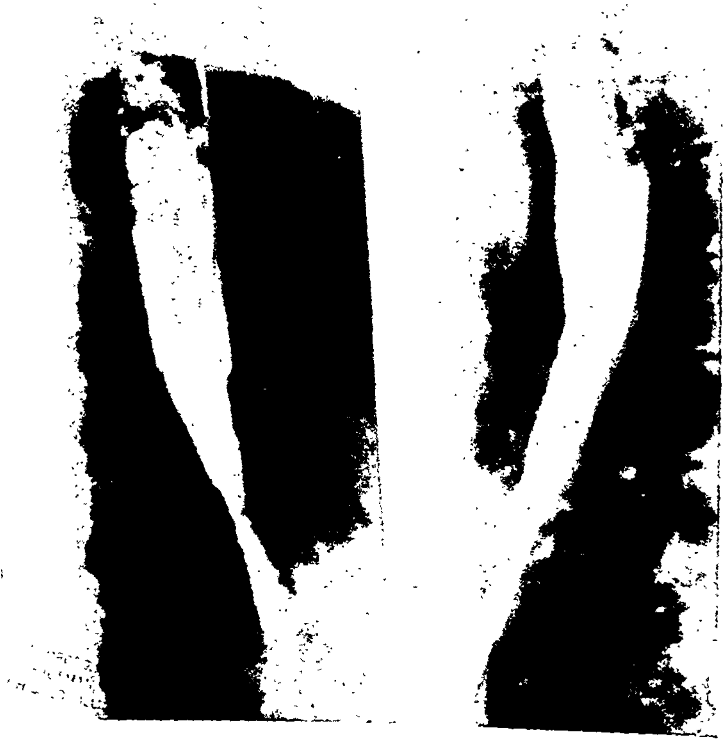

Fig. 1.—A constant filling defect is noted at the lower end of the esophagus.

CASE REPORT

The patient, J. M., a 51-year-old man, entered the hospital complaining of dysphagia, and loss of weight of eight months' duration. His past history revealed numerous episodes of gastric distress for twelve years. Six years prior to entrance he was operated upon for a perforated peptic ulcer. With the exception of having been a heavy drinker, his past history was otherwise noncontributory. Dysphagia began to develop six months prior to entrance; this became worse progressively until he was able to swallow only small sips of water.

Physical examination revealed an emaciated man weighing 101 pounds, who appeared very ill but not in acute pain. The lower left chest was dull to percussion; diminished breath sounds were noted both anteriorly and posteriorly over the base of the left side of the chest. With the exception of an upper right rectus incision the findings were essentially normal.

Roentgenography showed a marked filling defect involving the lower third of the esophagus with almost total obstruction (Fig. 1). In the region of the lower left lobe of the lung an opacity about the size of an orange was noted. Atelectasis could not be ruled out.

Esophagoscopy revealed an almost total obstruction of the esophageal lumen 42 cm. from the gingival border. This appeared edematous, red, and bled easily. Biopsy of this section showed inflammatory tissue. Following esophagoscopy the patient ran a febrile course which was controlled within a week by conservative therapy.

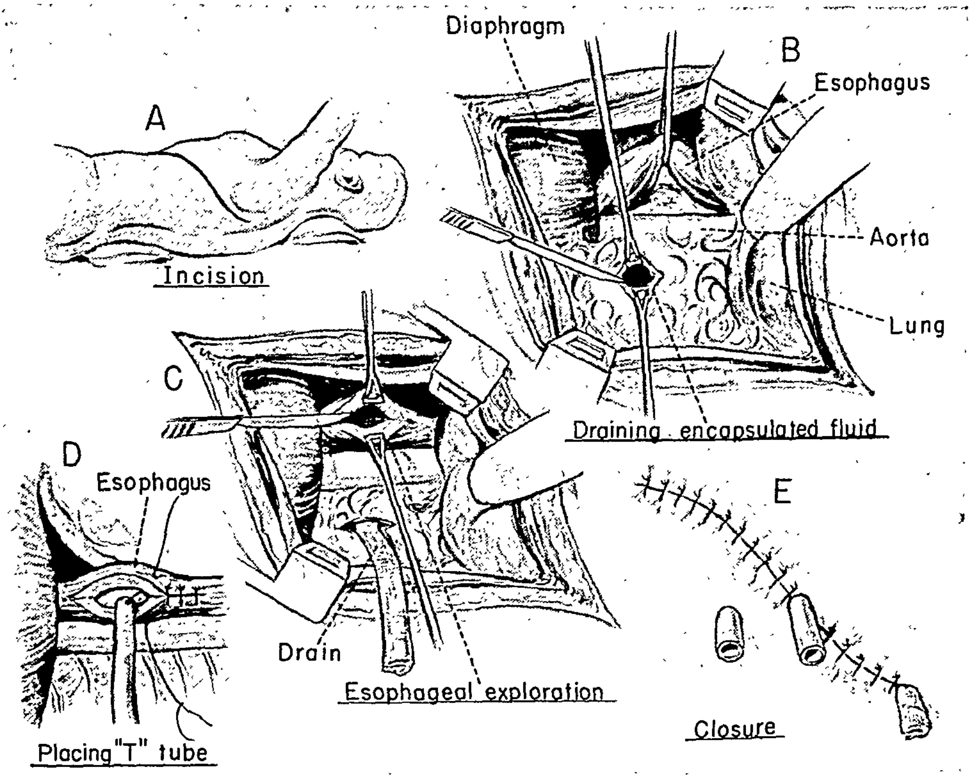

Fig. 2.—The operative procedure.

An exploratory thoracotomy was done ten days later, and an orange-sized mass in the left lower cavity was drained of turbid, flocculent, nonpurulent fluid (Fig. 2). The resulting cavity was emptied and packed completely, The lower lobe of the lung was firmly adherent to the left hemidiaphragm; the lower third of the esophagus was exposed and found to be scarred, rigid, and inelastic.

The involved portion of the esophagus was opened through a longitudinal incision to rule out a malignancy and to determine the size of the esophageal lumen. This disclosed a scarred and inflamed muscular layer and an almost obliterated lumen. No definite ulcer could be identified, although it was thought that at one time a small slow perforation occurred in this segment which leaked peripherally between the diaphragm and the lung, and accounted for the collection of turbid fluid found in the pleural cavity.

Resection was impossible because of the condition of the patient who was almost in extremis despite energetic and intensive preoperative care, contamination of the chest cavity, and the tremendous amount of fibrosis and edema which involved the lower end of the esophagus. It was thought inadvisable to attempt to close the esophagus over an indwelling tube since an esophageal leak semed inevitable. The possibility of chronic perforation could not be ruled out.

A large semi-rigid T-tube was placed into the esophagus through the incision and an external esophageal fistula was created. The T-tube was sutured into place and bilateral vagotomy was performed. Although the T-tube was large, the dysphagia disappeared rapidly.

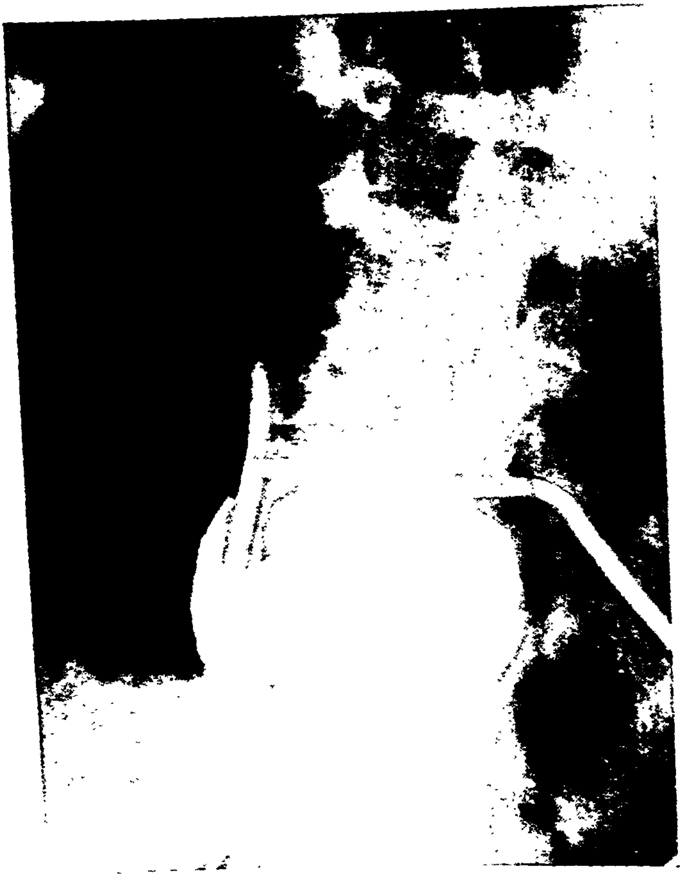

Fig. 3.—The T-tube shown in place in the lower end of the esophagus. It is noted that swallowed barium passes through the tube and around the tube, and readily fills the stomach. The limb of the tube which leads to the exterior is also filled with barium.

The patient was given fluids by mouth on the third postoperative day and a roentgenogram on the ninth postoperative day showed barium passing both into the stomach and out of the T-tube (Fig. 3). The T-tube remained in the esophagus for nine weeks, during which time the swallowed food which appeared externally out of the T-tube gradually diminished. Following removal of the T-tube the external opening closed within five days (Fig. 4), and the fistulous tract began to narrow and disappear.

Within the first six months of this procedure the patient gained 22 pounds, had a rather dilated esophageal lumen, no fistulous tract, and swallowed solid food with ease (Fig. 5).

This patient is being followed cautiously roentgenologically and will probably require an occasional dilatation; however, to date this has not been necessary.

SUMMARY

This report describes a case of stenosing esophagitis which resulted in almost complete obstruction of the lower end of the esophagus, and was associated with encapsulated fluid in the lower left chest, probably due to spillage from chronic perforation.

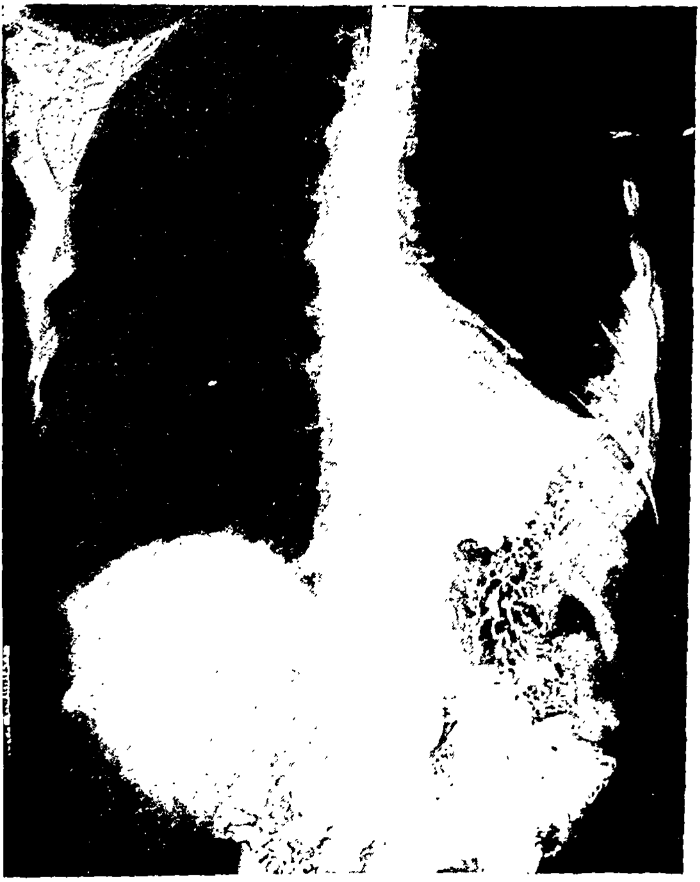

Fig. 4.

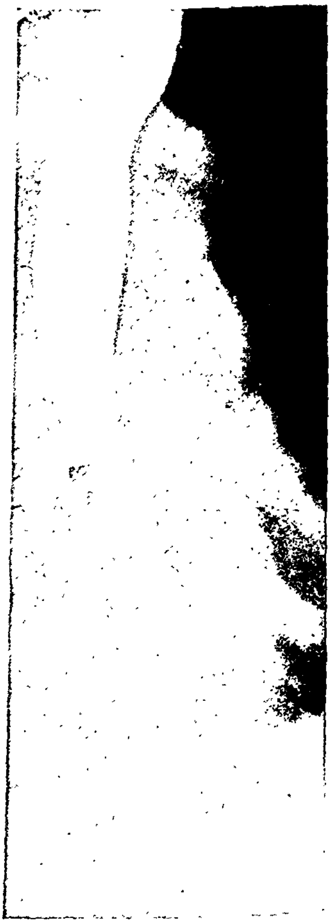

Fig. 5.

Fig. 4.—Five days following removal of the T-tube. Barium by mouth was given, and the narrowing of the fistulous tract is noted. The external opening of the tract is closed completely. The bulk of the barium has passed the now nonobstructed esophagus into the stomach.

Fig. 5.—Roentgenogram taken six months postoperatively. The barium passes freely into the stomach through a nonobstructed esophagus, and the fistulous tract has disappeared entirely.

Because of the patient's critical condition, practically in extremis, the contaminated pleural cavity, and the involvement of the esophageal wall, resection was considered impossible.

A T-tube was used to create a temporary external esophageal fistula.

To diminish hypersecretion and hyperacidity, bilateral vagotomy was performed.

At the present writing the patient feels perfectly well, his dysphagia has not recurred, and the fistula has closed.

After a perusal of the literature no report could be found of a procedure of this type being done for stenosing esophagitis with or without perforation.

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DISCUSSION

DR. RICHARD H. SWEET, Boston.—It seems to me that this procedure is exceedingly unsound because, if some of our less experienced associates attempt to treat the esophagus in this manner, I have the feeling they will get into all sorts of difficulties that a less experienced operator might be expected to encounter. Dr. Thorek's moving picture shows, to my satisfaction at least, that the lesion he encountered would lend itself readily to resection. If the wall appeared to be involved in so much inflammatory thickening that it might not hold sutures satisfactorily, all that would be necessary would be to follow the esophagus upward to a proper level just below the aortic arch or above the arch, and then pull the stomach up and do an anastomosis.

I am quite certain that I have performed an esophagectomy in many cases in which there was esophagitis quite as bad as, if not worse than, in this case.

DR. THOREK (closing).—Just a word to thank Dr. Sweet for his remarks. I can state only that one must take sincerely the comments and criticisms of one so versed in esophageal surgery.

OBSERVATIONS AS TO THE ETIOLOGY AND TREATMENT OF ACHALASIA OF THE ESOPHAGUS*

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THE physiopathology of achalasia of the esophagus is not clearly understood even though the first recorded case was in 1672[1] and theories as to etiology have been propounded in the literature ever since. This state of knowledge results from the differences of opinion regarding the anatomic and physiologic features of the esophagus and cardia of the stomach under both normal and diseased conditions.

The earlier investigators stressed the cardia of the stomach as the site responsible for this condition. In 1882, von Mikulicz[2] popularized the theory of a true spasm of a cardiac sphincter as being the etiology of a mega-esophagus. Einhorn,[3] in 1888, and later Hurst,[4] in 1914, propounded the theory of "failure of relaxation" of the cardiac sphincter, and termed this condition achalasia. Since this time observers have differed as to the presence of a cardiac sphincter.

At the present it is generally accepted that a cardiac sphincter as such does not exist but that there is a closing mechanism present at the cardia produced by the slinglike action of the inner and oblique muscle layers of the stomach partially encircling the cardia. Experimental observations suggest that this mechanism, in conjunction with the septum between the esophagus and cardia on the left side, is probably more important in preventing regurgitation than it is in participating in the act of deglutition. Both experimental and postmortem observations show that the cardia appears to be in a state of relaxation except when the stomach is distended. Inspection of the cardia during the performance of a cardioplasty as well as microscopic examination of sections taken from the cardia of patients with achalasia elicit no abnormal findings. The junction between the esophagus and cardia also appears normal. There is, however, generalized hypertrophy of the esophageal musculature and particularly the circular muscle of the terminal esophagus, as will be described later.

Extra esophageal lesions such as deformity of the "liver tunnel" with kinking of the esophagus,[5] periesophageal fibrosis,[6] incoordination of the diaphragmatic crura,[7] phrenospasm,[8] and failure of the diaphragmatic pinchcock to open during the process of deglutition[8] have all been proposed as causing the obstruction. None of these theories have been substantiated.

Next, various neurogenic theories were presented. These were based on microscopic and experimental observations. Rake[9] (1927) described absence of and degenerative changes in the cells of Auerbach's plexus in the lower

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*Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

esophagus in patients having achalasia. This finding deserves more emphasis than generally accorded it. As illustrated in Fig. 1, Auerbach's plexus, as well as the myenteric nerve fibers, may appear essentially normal. In other cases the ganglia cells may be difficult or impossible to find, or may show round cell infiltration, fibrosis, and degeneration. The scarcity of ganglion cells might be interpreted as being the result of the esophageal dilatation and not the result of the loss of ganglia if it were not for the fact that the contracted terminal segment of the esophagus appears to be more lacking in ganglion cells than in the dilated portions. There appears to be a correlation between the scarcity of ganglion cells and the degree of achalasia. The photomicrograph illustrated in Fig. 1 demonstrates the presence of ganglia cells showing some granulation suggestive of degeneration in a patient with a moderate degree of achalasia as demonstrated by the esophagram in Fig. 2, as compared to the photomicrograph in Fig. 3 where multiple serial sections failed to show any ganglia cells even though there were numerous nerve bundles. This patient had the most severe type of achalasia seen. The entire right hemithorax was filled with this dilated esophagus, as demonstrated in Fig. 4.

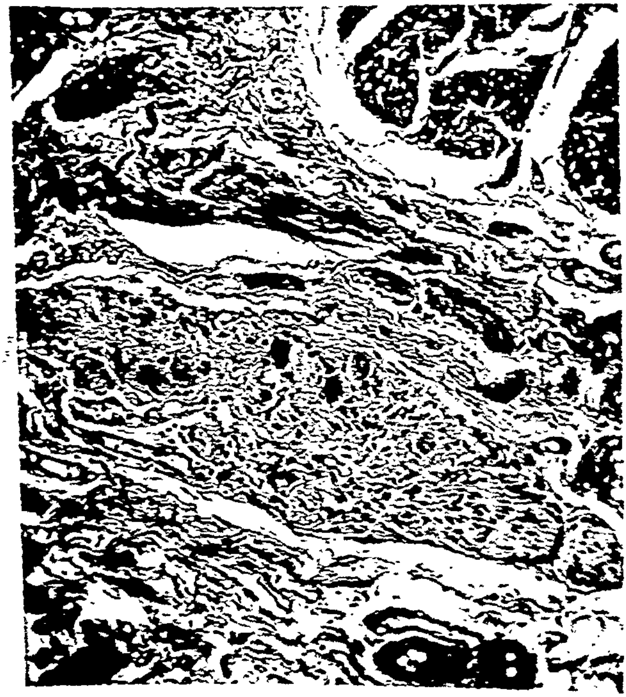


Fig. 1.—Photomicrograph of Auerbach's plexus showing some degeneration of the ganglion cells in a patient with moderate degree of achalasia (Fig. 2). (X120.)

The peristaltic reflex and muscular action of the distal esophageal segment are under the control of the ganglion cells. When there is degeneration, reduction, or absence of these ganglion cells, or when the myenteric plexus is destroyed experimentally as demonstrated by Tamiya and Tamura[10] there is a preponderance of sympathetic control; muscular coordination is impossible; and the circular fibers become contracted and hypertrophied to the degree of causing esophageal obstruction. As the result of this obstruction secondary esophageal dilatation and hypertrophy develop above. When the pressure within the esophagus builds up, or if the patient voluntarily increases his intrathoracic pressure by the Valsalva maneuver food or barium can be pushed into the stomach. Barium may also be demonstrated fluoroscopically to enter the stomach during the midrespiratory phase, as will be described later. The foregoing findings are similar to those observed in megacolon.[11, 12] The parallelism between these two conditions is of interest.

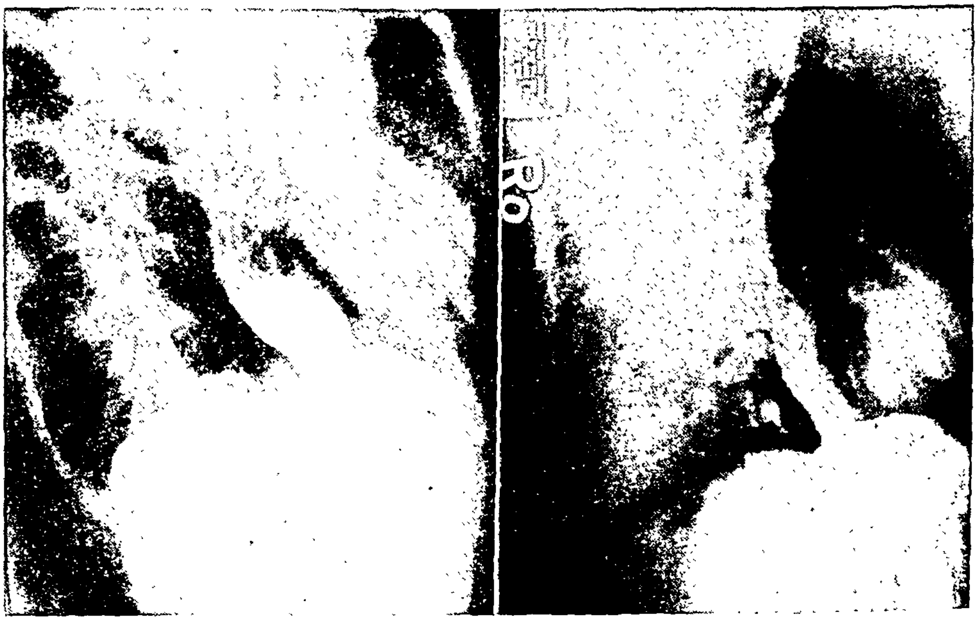


Fig. 2.—Esophagus demonstrating achalasia of esophagus before and after Heineke-Mikulicz type of cardioplasty.

When the obstruction is relieved by dilatation or operation, peristalsis may be restored and the esophagus may reduce in size if the degree of dilatation has not been too extensive, which suggests that the ganglia and nerve fibers of the upper esophagus are capable of function. If this condition were due to vagal nerve damage one would not expect peristalsis to be re-established. A stage in the diseased process may be reached, however, where irreversible damage to the esophageal wall and nerves may have occurred. The pathologic findings presented consequently are dependent upon the stage of development of the achalasia. The more extensive findings are usually found in the greately dilated sigmoid type of esophagus.

Knight[13] and Knight and Adamson[14] presented experimental evidence to show that the condition can be simulated experimentally in cats by sectioning the vagus nerves. They also showed that this experimentally produced mega-esophagus can be prevented by simultaneous section of the sympathetic fibers, or when produced can be relieved later by this procedure. This led to the view that inherent disease in the vagus nerves might be responsible.

Stimulation of the vagus nerves at the time of operation causes contraction of the esophageal musculature and relaxation of the sphincter in the milder cases of achalasia. No significant muscular response is seen following vagal stimulation in the far-advanced case, which suggests that the vagal dysfunction may be a secondary manifestation in the far-advanced case, or, of course, may be without effect because of possible loss of underlying ganglion control. This finding is in keeping with fluoroscopic evidence of increased primary and secondary peristalsis in the upper and mid-esophagus in patients with milder degrees of achalasia, as compared to the complete loss of peristalsis and loss of esophageal tone in the far-advanced stage. A condition simulating achalasia may result from a chronic mediastinitis or mediastinal lymphadenopathy with involvement of the vagus nerves.

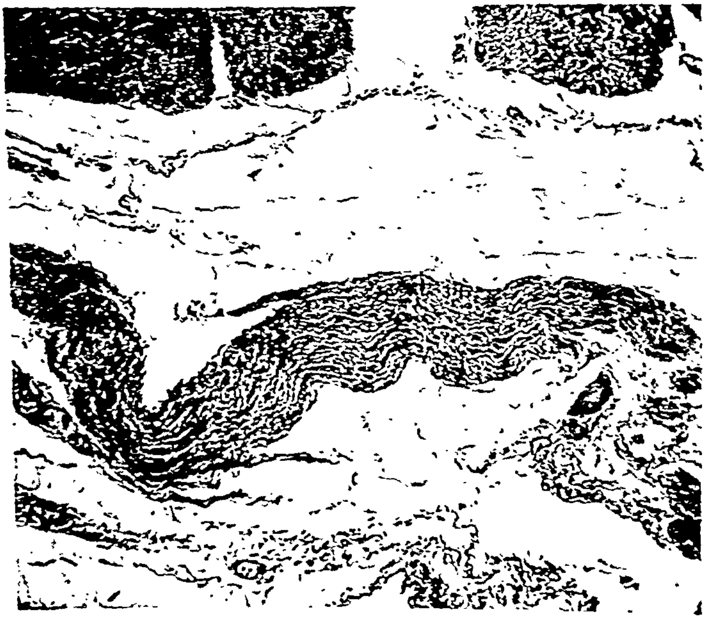


Fig. 3.—Photomicrograph of Auerbach's plexus demonstrating absence of ganglion cells in severe type of achalasia as demonstrated in Fig. 4. (X120.)

Vagal denervation associated with esophageal mobilization causes dilatation of the esophagus and contraction at its terminal region. Also transthoracic vagotomy for peptic ulceration is occasionally followed by transitory terminal esophageal spasm which persists for three to five weeks before the esophagus recovers its own automaticity.

The terminal esophagus in some patients with achalasia can be demonstrated fluoroscopically to dilate like a sphincter under the influence of amyl nitrite and ergotamine (Fig. 5). No such action is noted in other regions of the esophagus. This sphincterlike mechanism can be demonstrated by surgical dissection to be hypertrophied circular muscle fibers confined to the distal 2 or 3 cm. of the terminal esophagus; above this site the circular and oblique fibers are not hypertrophied to the same extent. The dilating action of amyl nitrite on the sphincterlike mechanism of the hypertrophied circular muscle in the terminal esophagus has also been observed in some cases of achalasia at the time of operative exposure.

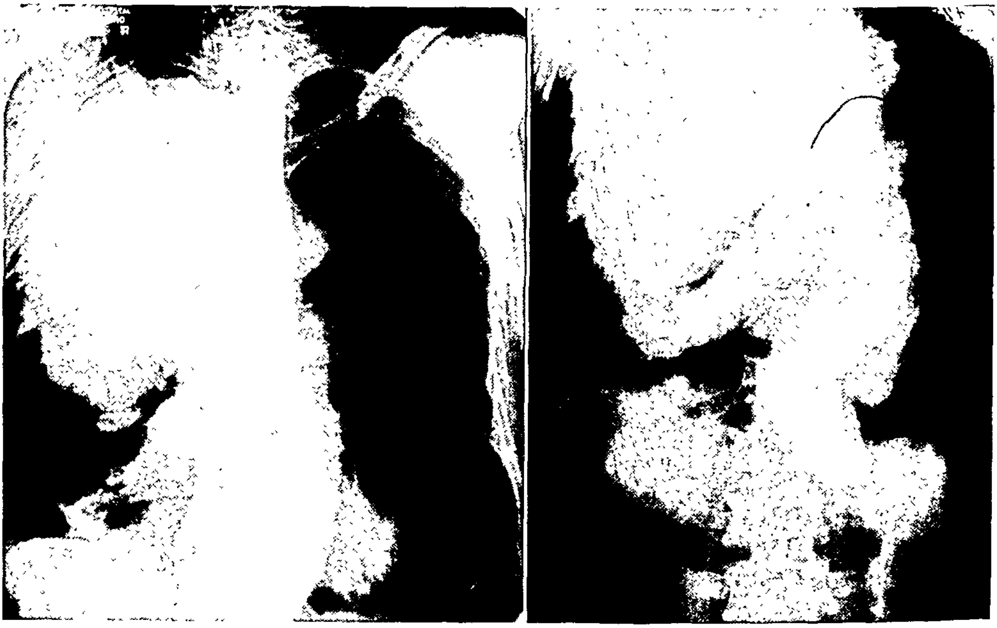


Fig. 4.—Esophagram demonstrating markedly dilated esophagus filling entire right hemithorax.

Why the circular muscle layer in the distal esophagus should hypertrophy to the extent of causing functional esophageal obstruction with secondary esophageal dilatation above and not respond in the normal manner to either the primary or secondary esophageal peristaltic waves is not known, but appears to be the result of lack of ganglion control and coordination. Etzel[15] has suggested a vitamin B deficiency as a possible cause of the nerve damage when degeneration of the ganglion cells is seen. In support of this theory are other types of so-called "functional dysphagia" which respond to vitamin B therapy. The reduction in number or complete absence of ganglia cells in the terminal esophagus may be on a congenital or developmental basis. Others[16] have suggested psychogenic causes. Those emotional problems seen in these patients are more likely due to their esophageal dysfunction than to primary psychic disorders, for they soon disappear when normal eating is restored. One might say, however, that the majority of these patients tend to be the apprehensive introspective type of individual.

It is felt that a review of certain of the anatomic features of the esophagus and cardia would be pertinent to this discussion. The esophagus has a well-developed outer longitudinal muscle layer and a less well-developed inner muscle. The longitudinal muscle extends from the cricoid cartilage and cricopharyngeus muscle above to insert below in an interlacing manner into the middle and outer muscle layers of the cardia. Muscle fibers from the aorta, bronchi, and pleura attach themselves to the longitudinal muscle throughout its course. The upper one-fifth is striated, and the remainder is smooth muscle.

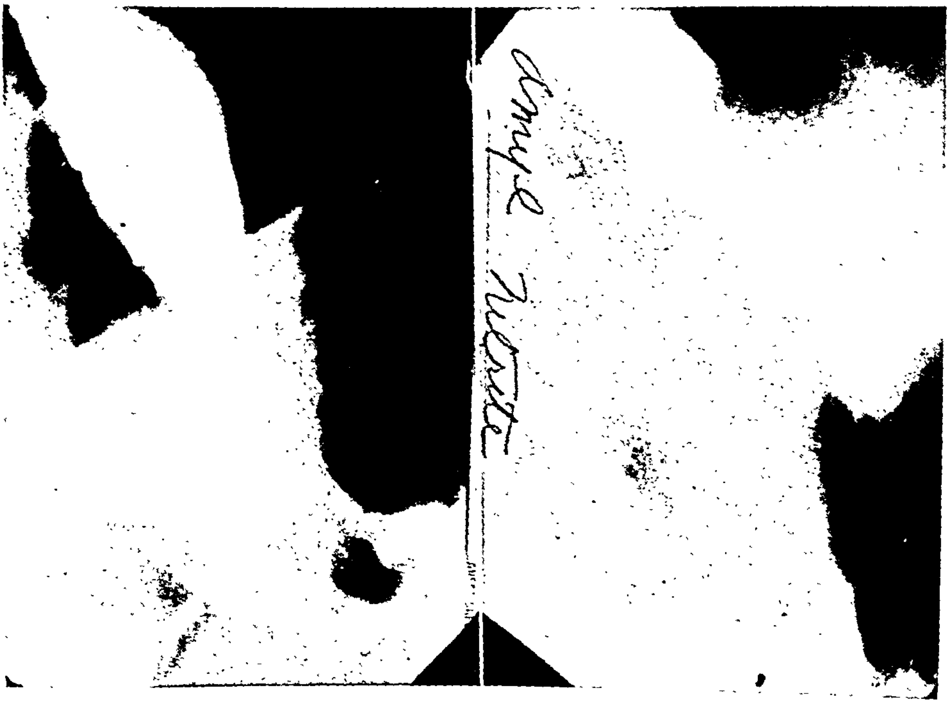


Fig. 5.—Roentgen evidence of dilating effect of amyl nitrite on circular muscles of terminal esophagus in patient with achalasia.

According to Lerche,[17] Laimer described the inner muscle layer of the esophagus as commencing in the upper end independently of any other group of muscle fibers. The arrangement of these fibers varies in the different segments of the esophagus. These muscle fibers run in an elliptical direction around both sides of the esophagus and downward in the upper third of the esophagus. A small segment of the mid-esophagus about 1 cm. in length has circular fibers, below which they again run in an elliptical course in a downward direction toward the posterior aspect of the esophagus. In the lower esophageal third, according to these authors, the muscle fibers wind about the esophagus in a spiral manner. There are also occasional spiral fibers throughout the other segments. These spiral fibers appear to be responsible for the tertiary contractions and "curling" occasionally noted fluoroscopically. The inner muscle layer of the esophagus continues in the inner and middle muscle layers of the cardia and stomach. Less often there are elliptical or circular fibers in the esophagus just above the cardia. Laimer and Lerche also described from anatomic studies a circumscribed thickening of the inner muscle layer approximately 1 to 3 cm. above the diaphragmatic hiatus which they referred to as the lower physiological constriction or the inferior esophageal sphincter. Deeper longitudinal fibers from this sphincter insert into the longitudinal layer and have a dilating action on the sphincter.

According to the above description these separate muscle fasciculi appear quite apparent but unless they are carefully searched for the variations of the inner muscle layer and particularly the sphincterlike action of the circular and elliptical fibers in the terminal portion of the esophagus may not be too obvious.

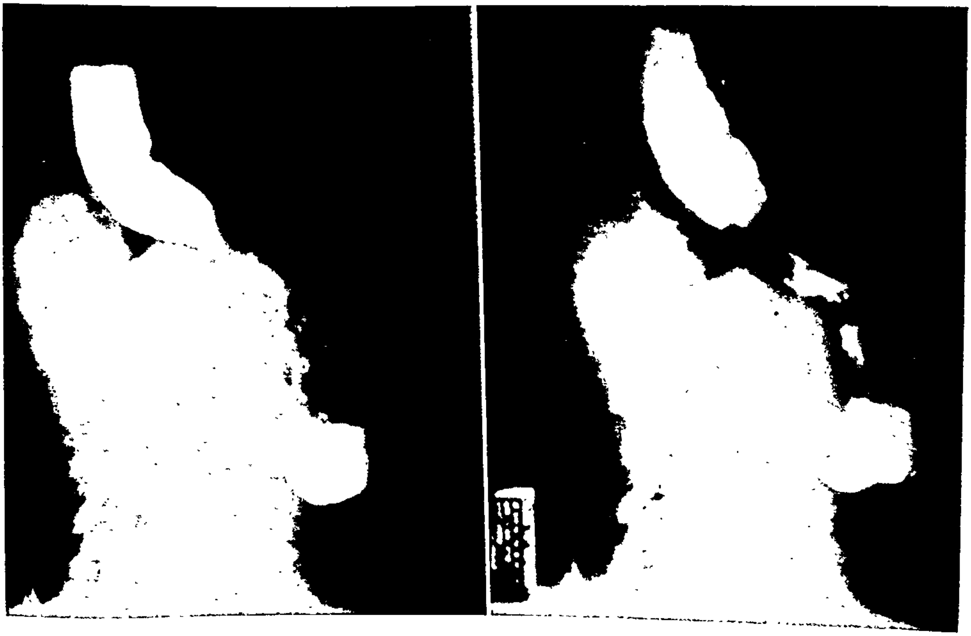
As contrasted to the normal, anatomic and surgical dissections reveal the inferior esophageal constriction in patients having achalasia to be very definite and composed of markedly hypertrophied circular and elliptical muscle fibers extending from the esophagogastric junction upward for 2 to 5 centimeters. This area corresponds to the contracted distal segment of the affected esophagus.

The diaphragmatico-esophageal ligament may also play a role in both normal and abnormal deglutition. This ligament was first described by Laimer[18] in 1883. It arises from the fascia on the under surface of the diaphragm. At the hiatus it divides into an ascending part that extends upward and attaches to the longitudinal and circular muscle fibers of the lower esophagus at the site of the inferior esophageal constriction or sphincter, and a descending limb that attaches to the cardia. This ligament is the antagonist of the longitudinal muscle of the esophagus and prevents the esophagus from pulling the stomach into the chest during swallowing. There is considerable variation in the thickness of this ligament. When it is stretched or atrophic it may be one of the factors responsible for the development of hiatal hernias. In some patients with achalasia the ascending limb of this ligament has been taut and hypertrophied, and has appeared to be playing a role in the causation of the esophageal constriction. In one patient, not until this ligament was entirely sectioned at a secondary operation was the esophageal obstruction relieved.

It can be demonstrated fluoroscopically that the point of obstruction in patients with achalasia is definitely related to the stress upon either the ascending or descending limb of this ligament. As illustrated in Fig. 6 on deep inspiration the diaphragm descends, the ascending limb becomes taut and the point of esophageal obstruction is several centimeters above the diaphragm. On expiration the diaphragm ascends, the descending limb becomes taut, and the point of obstruction is then at the cardial junction.

The diversity of opinion regarding the etiology of cardiospasm has also been reflected in the multiplicity of measures employed for its relief. Ochsner and DeBakey[19] have ably covered the literature on this subject and the reader is referred to this review. Only those therapeutic measures designed to enlarge the inferior esophageal segment and cardia have survived the test of time. Other measures attacking the esophagus proper, the diaphragm, or the nerve control have failed in their expectations. The techniques most commonly employed have little specificity as to the exact site of benefit and are of little value in contributing knowledge as to either this site or to the nature of the obstruction.

Patients with mild to moderate degrees of achalasia frequently obtain considerable relief of symptoms and occasional reduction in the size of the esophagus following mechanical dilatation. This dilating effect, however, is employed throughout the terminal esophagus and cardia so it is impossible to say with certainty exactly what had to be dilated.



A. B.

Fig. 6.—Esophagrams demonstrating that point of esophageal obstruction related to the stress upon either the descending or ascending limb of the diaphragmaticoesophageal ligament. *A*, expiration: *B*, inspiration.

In other patients with more severe degrees of achalasia dilatation may not be possible because of the tortuosity of the esophagus or the benefit is so temporary as to be without value. These patients can be benefited by operative procedures. These procedures fall largely into two classes, the cardioplasty of either the Finney or Heineke-Mikulicz type performed either from above or below the diaphragm and the cardiomyotomy somewhat modified from that originally described by Heller.

The majority of those surgeons employing the Finney type of cardioplasty have stated that there is more symptomatic improvement than there is objective evidence of relief of the obstruction. The Heineke-Mikulicz procedure performed transthoracically as described in a previous communication[20] not only gives symptomatic relief but objective evidence of marked reduction in the size of the dilated esophagus. In many patients return of peristalsis and muscular tonicity is evident following relief of the obstruction. Again as in esophageal dilatation, these surgical procedures are not specific as to their site of benefit. These techniques necessitate an incision through the lower esophagus, esophagogastric junction, and cardia so that all structures are incised. These operations are usually followed by both functional benefit and also evidence of reduction in the size of the esophagus if the new stoma is made sufficiently large.

Though relief of esophageal obstruction is obtained and the patient is satisfied with his ability to swallow, the incidence of regurgitation esophagitis and gastritis with secondary bleeding and anemia is so high as to make this operative technique less attractive. Nine of thirty-two (28 per cent) patients having a Heineke-Mikulicz or Finney type of cardioplasty have had evidence of esophageal bleeding and anemia. In four of the patients bleeding was severe. All of the nine patients have to follow a carefully regulated ulcer regimen. Gastric analysis has not shown hyperchlorhydria. Two of the patients who have bled most severely showed a persistent achlorhydria. In one of these patients it was necessary to do a secondary gastric resection to control the ulceration and bleeding. This particular patient originally had a Finney type of cardioplasty performed transabdominally. Vagus section at the time of the cardioplasty appears to be of no value in the prevention of this ulceration and bleeding and may give further symptoms secondary to possible pylorospasm.

Because of this incidence of bleeding associated with the cardioplasty technique, it was felt that a better long-range result might be obtained following resection of the lower esophagus along with the cardia and fundus of the stomach. Of three patients so treated and followed for two years, all three have been able to eat satisfactorily, but one follows quite closely an ulcer regimen because of the occurrence of a regurgitation esophagitis and hemorrhage one month postoperatively. There has been no evident subsequent bleeding.

Though this technique appears to be satisfactory, the results are no more gratifying than what can be obtained by a modified Heller operation which is of considerably less operative magnitude. During the past year six patients ranging from 9 to 53 years of age have been operated upon by the modified Heller technique. There has been a satisfactory functional response in all, with a corresponding gain in weight. There has been no evidence of postoperative esophagitis and bleeding in these patients as yet. It is felt that by preservation of the esophagogastric junction in the technique to be described accounts for the absence of regurgitation and prevents the development of an esophagitis.

It is of interest that in the careful performance of the modified Heller technique, a markedly hypertrophied circular muscle at the site of the inferior esophageal sphincter described by Laimer and Lerche has appeared sufficiently contracted as to be responsible for the esophageal obstruction.

The Heller operation as originally performed consisted of exposure of the stomach and cardia through a laparotomy incision, mobilization of the terminal esophagus and cardia and parallel longitudinal incisions of the musculature down to the mucosa anteriorly and posteriorly.

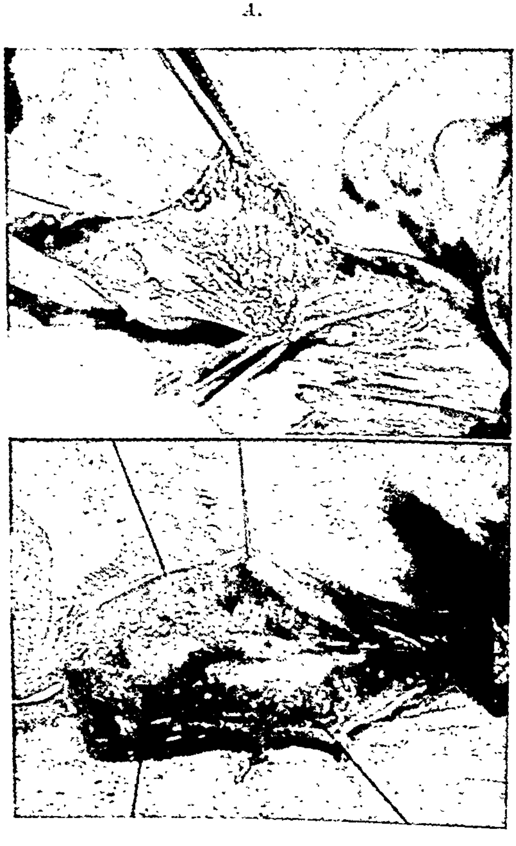


Fig. 7.—Surgical exposure demonstrating hypertrophied circular muscle fibers in terminal esophageal segment before *A*, and after *B*, severing these fibers and allowing the mucous membrane to bulge.

The technique as now performed in these patients through a thoracotomy incision consists of separating the longitudinal muscle of the terminal esophagus and isolating the hypertrophied circular muscle. This has been approximately two times the thickness of the longitudinal muscle. This hypertrophied circular muscle which extends upward from the esophagogastric junction for 1 to 5 cm. is then severed allowing the submucosa to bulge (Fig. 7). Heretofore the esophageal musculature has been divided down to the submucosa without differentiation into two layers.

The results of transecting this sphincterlike hypertrophied circular muscle has been gratifying in all patients so treated even though there has not been as complete a reduction in the size of the esophagus as usually noted following a Heineke-Mikulicz cardioplasty (Fig. 8).

As experience is gained in the treatment of this condition it may be found that the type of operative procedure employed in those patients not responding to dilatation will depend largely on the pathologic features of the presenting esophagus. In extensively dilated esophagi with loss of peristalsis the most satisfactory procedure may still be a cardioplasty or a partial esophagogastric resection where a large stoma can be made which cannot possibly allow a retention of food. In the less extensive cases the modified Heller operation will suffice. From experience so far attained it appears that this latter operation, however, when correctly executed, will fulfill all of the surgical requirements and not be associated with unfortunate postoperative complications such as regurgitant esophagitis.

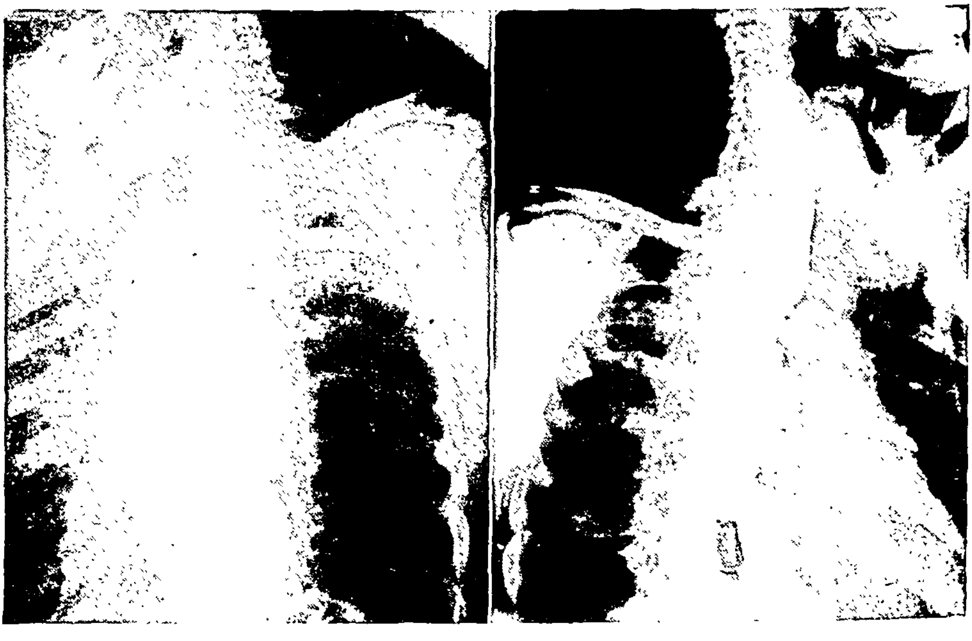

A. B.

Fig. 8.—Esophagrams demonstrating achalasia of esophagus before and after modified Heller cardiomyotomy.

In conclusion, there is evidence to show that when there is degeneration, reduction, or absence of ganglion cells in Auerbach's plexus, a preponderance of sympathetic control develops; muscular coordination is impossible; and the circular fibers become contracted and hypertrophied to the degree of causing esophageal obstruction with secondary esophageal dilatation and hypertrophy above. This terminal esophageal obstruction can be adequately relieved by dilatation or by severing the hypertrophied circular muscle in the terminal esophageal constriction or sphincter.

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DISCUSSION OF "THE TREATMENT OF CARDIOSPASM: ANALYSIS OF A TWELVE-YEAR EXPERIENCE," BY DR. ARTHUR M. OLSEN, DR. STUART W. HARRINGTON, DR. HERMAN J. MOERSCH, AND DR. HOWARD A. ANDERSEN, AND "OBSERVATIONS AS TO THE ETIOLOGY AND TREATMENT OF ACHALASIA OF THE ESOPHAGUS," BY DR. EARLE B. KAY

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—I had the opportunity to discuss the subject of mega-esophagus at the recent meeting of the American Surgical Association in Washington. However, because of the more complex nature of this audience I welcome the opportunity to say something about it here. In his splendid presentation of his subject, my colleague from Rochester, Minnesota, Dr. Olsen stated that 25 per cent of his patients were untraced. It is probably well to remind ourselves that, it is easier to trace the satisfied patient than the dissatisfied patient.

Mikulicz gave this entity its name, in 1882, when he called it cardiospasm. I believe some light is coming into our understanding of the nature of the disturbance. I prefer to call this entity dystonia rather than cardiospasm or achalasia, because the disorder has two components, namely: hypertonus of the distal segment and atony of the more proximal unstriated musculature of the esophagus.

The objective of conservative management is a bloodless myotomy of the hypertonic segment. In Dr. Olsen's traced cases that objective apparently was achieved in 60 per cent of instances in one hydrostatic dilation. My plea would be for earlier resort to operative myotomy if one or two attempts at bloodless myotomy fails. In many of our Outpatient Clinics, patients are coming back repeatedly over a period of years for dilatation. In other words, some of these patients are denied effective treatment and are subjected to periodic dilatations of the hypertonic esophageal segment with bougies. Cures of esophageal dystonia by dilatation with bougies must be infrequent. Distrust of, or dissatisfaction with conventional surgical treatment of the management of mega-esophagus is probably, in part, responsible for this practice. Now, that the desiderata of satisfactory surgical management of esophageal dystonia, are becoming evident, there is no need to temporize indefinitely with bougie and hydrostatic attempts at esophageal dilatation. Moreover, as Dr. Olsen has indicated, hydrostatic dilatations also carries hazard.

The honeymoon of many an operative procedure for esophageal dystonia is over, and the last of some of the proopsed operations, let us hope, has been done. I speak especially of the Wendel and the Heyrovsky-Gröndahl operations for cardiospasm. Both these operations, it is now known, invite esophagitis. The two operations which correct the condition without being followed by esophagitis are the Heller extra-mucosal myotomy and the operation described by me in 1949, in which the tortuous distal end of the esophagus is excised together with the entire acid secreting area of the stomach. That there should be such an important difference between the results of the Wendel procedure of open cardioplasty and the Heller extra-mucosal myotomy is very interesting. It had been believed, of course, that the only important condition to be met was to provide satisfactory emptying of the dilated esophagus. It now turns out that the Wendel procedure encourages regurgitation of acid-peptic juice into the esophagus, whereas the Heller operation apparently does not. Barrett of London has presented critical evidence on this point. (Proceedings of the Royal Society of Medicine, June, 1950, Section on Surgery.)

It is for the tortuous, baglike esophagus that I have done the larger operative procedure, straightening out the esophagus and excising its tortuous distal segment, together with the hypertonic segment and the entire acid secreting area of the stomach. A coincidental Ramstedt procedure is also done on the pylorus to insure satisfactory gastric emptying after excision of the vagi nerves, which of necessity attends the procedure.

Demonstration of the sensitivity of the esophagus to acid-peptic juice was, I think, a startling observation to all of us. Most of us had been unaware of it. It had been reported now and then. The paper by Pringle and Teacher of Glasgow (1919) is particularly good. Yet, pathologists had continued to insist that the evidences of esophagitis, and even perforation of the esophagus occasionally observed at post mortem—that these were essentially agonal or post mortem changes owing to softening of the esophageal wall occasioned by the reflux of acid-peptic juice into the esophagus during the agonal state. We need to remind ourselves periodically that there are no Delphic oracles in medicine. Observations in the experimental laboratory have shown that the esophagus is more sensitive to acid-peptic juice than is any juxta-gastric lying mucosa. The cat's esophagus may perforate within twenty minutes, attending the dripping of gastric juice into it, obtained from an isolated canine gastric pouch; the cat's esophagus is by no means that sensitive to injury by hydrochloric acid of the same pH.

In doing the Heller myotomy, I like to make a short transverse gastrotomy incision in the upper stomach. Under visual and tactile control, with the finger inserted into the esophagus, the division of the muscular fibers of the hypertonic segment is readily done, without risk of injury to the mucosa. Moreover, this incision should be adequately long. Ten to twelve centimeters is not too long. Some of the reported failures, in the Heller myotomy, are undoubtedly owing to the myotomy being inadequate. I like to assess whether or not the deepest fibers of the circular muscle have been divided by the aid of a large magnifying glass. The median extrapleural sternotomy incision gives direct and easy access for performance of either the Heller myotomy or the more formidable procedure of excision of the acid secreting area of the stomach, together with the tortuous lower segment of the esophagus.

I believe there is a functional sphincter at the esophagogastric juncture. It never acquires the proportions of a tumor as in hypertrophic pyloric stenosis; I do not believe the sphincteric action is owing to any diaphragmatic attachment. When one inserts his finger at operation through the esophagogastric juncture he can feel the constriction, just as when the finger is inserted through the pylorus. Maybe, some day, the anatomists will recognize the presence of a sphincter at the gastric inlet.

In the discussion of this subject at the recent meeting of the American Surgical Association, Dr. Evarts Graham stated that he had been performing myotomy operations on the rectosigmoidal area for megacolon. I am inclined to believe that mega-esophagus, congenital hypertrophic pyloric stenosis, and megacolon may be somewhat related entities.

Finally, I want to say that I do not believe that diffuse esophageal spasm is mega-esophagus at all; the spastic proximal reaches of the esophagus are hypertonic and not atonic as in the usual esophageal ectasia. If a patient has had dysphagia for several years and there is no atony or ectasis, the patient does not have so-called cardiospasm or mega-esophagus. Understanding is beginning to come into the realm of the misunderstood esophageal dysphagias. Idiopathic stricture and spontaneous perforation of the esophagus are manifestations of acid-peptic disease, owing to reflux of gastric juice. Mega-esophagus is a functional disorder, probably owing to absence in the esophageal wall of nerve cells in the terminal plexuses of the parasympathetic nervous system. Neither sympathectomy nor vagotomy have any place in the treatment of mega-esophagus. When an attempt or two at effecting a bloodless myotomy by hydrostatic dilatation fails, operation is in order. And satisfactory operations are now available with which to treat the disorder, as well as to thwart the complication of esophagitis which has been the nemesis of many operative procedures suggested for the relief of esophageal dystonia.

DR. RICHARD H. Sweet, Boston, Mass.: I must say that I have enjoyed these two presentations and the discussion very much. The sum total of it all is that I am in a far greater state of confusion now than I was when I came here; I might add that that is considerable.

In the first place, I have a very definite belief that it would be advantageous to us to abandon the term "cardiospasm." I personally have not observed cardiospasm in any of these cases, although perhaps the trouble is that I am not seeing the same type of patient. I do not find that the difficulty, the objective finding, at any rate, is at the *cardia* at all. This has been very striking to me. To be sure, my own experience is limited, having operated on only thirty-two cases, but I have observed that the abnormal region is not at the cardia but just proximal to it. This observation holds for all the cases I have actually exposed surgically through the thorax with good exposure and visualization of the lower end of the esophagus. It is very striking that the lower end, sometimes for a distance of several centimeters, sometimes only one or two, is strikingly narrow; it is hardly the size of an infant's esophagus at that point; its wall is thin. I cannot find that sphincter that Dr. Wangensteen speaks about although I have looked for it, and I cannot find the hypertrophy of that region that Dr. Kay described. It always looks to me like a *small* atrophic segment and yet proximal to that we see the enormous dilated hypertrophied boggy tortuous esophagus.

With regard to the Auerbach's plexus, in the six patients on whom I have carried out a resection, and two others in whom a secondary resection was done, making eight in all, we have searched for evidences of the ganglion cells and have found them diminished or absent, not only in the small atrophic-looking distal segment, but also as far up on the esophagus as we have gone. This finding has been pointed out before by others. Our experience with dilatation has been similar to that of this marvelous series of Dr. Olsen. From the standpoint of surgical treatment, I thought I might rehearse for you my experience with thirty-two cases. There were six primary resections. All had 100 per cent relief of dysphagia. But I find on review of the cases that there were two with signs or symptoms of esophagitis: one had some hemorrhage. Of the twenty-six on whom I performed esophagoplasty, by the method of longitudinal incision and circumferential closure, which I think must be rather an extensive one as Dr. Wangensteen pointed out, four showed some signs or symptoms of esophagitis; one of those had slight hemorrhage, and two had massive hemorrhage requiring reoperation and radical resection. So we have seen three cases of hemorrhage in the twenty-six patients.

I am not yet prepared to give this procedure up. I think if we surgeons had given up subtotal gastrectomy for duodenal ulcer in the early days of its use we would have come to regret it. There are certain other operations also which have gone through a phase of experimental development. I feel, therefore, that in the limited number of cases of mega-esophagus, idiopathic dilatation, or whatever we chose to call it, where surgery may be required, that it is up to some of us to try to improve the technique. I think that one error I have made in the past has been to allow the stomach to rise above the diaphragm. I think we shoudl maintain the integrity of the diaphragm and keep the stomach down, because some of our cases have had gastritis as well as esophagitis. It seems to me, in conclusion, that the problem is not quite as easily solved as it would seem to be on superficial thought.

DR. JOHN H. GARLOCK, New York.—We, too, have had our troubles with this condition, and I would like to report our experience with twenty-one patients who were operated upon. Our gastroenterologists have referred for surgery only those patients who had a megoesophagus with reduplication of the organ rather than the straight conical variety without elongation of the organ. Our experience, therefore, has been limited almost entirely to the complicated variety.

(Slide) As you will see, we have operated on twenty-one patients and have confined the operative procedure to two varieties; the Heyrovsky-Finney or Gröndahl operation was done on ten patients and the transthoracic lateral anastomosis was done on eleven patients. The results are indicated. There were two cases of bleeding in the first group and one in the second group secondary to a peptic esophagitis. It has been our experience that the longer these patients are followed, the greater will be the incidence of esophagitis and subsequent hemorrhage, and that has been the difficult problem so far as we are concerned. There is very little disturbance in the act of deglutition after an adequate anastomosis has been made, but the regurgitation of acid juices from the stomach, or whatever other factor causes that particular circumstance, the subsequent development of esophagitis and bleeding in an increasing number of cases is the important item in appraising the results of the various operations.

I do not think that the problem is as simple as Dr. Wangensteen has indicated in his discussion. This opinion is based on our experience with a number of patients in whom the physiologic gastric studies were so bizarre as to make one wonder about the role of regurgitated gastric contents in the subsequent development of esophagitis. I would like to cite briefly two such cases. There was one patient who had in 1948 in another hospital a Heineke-Mikulicz cardioplasty by a combined abdominothoracic incision. He developed marked esophagitis and hemorrhages. He was operated upon again because the roentgenogram suggested strongly the presence of a duodenal ulcer. The esophagoscopy at that time was negative. He had a high subtotal resection for a scarred area in the first portion of the duodenum which was thought to be a healed ulcer. The severe hemorrhages continued. Esophagoscopy then revealed that he had an extensive peptic esophagitis with ulceration. In 1949, a jejunostomy was done to put the esophagus at rest, and six months later a transthoracic lateral esophagogastrostomy was done. Since that time he has been perfectly well and, in spite of the fact that he had a lateral anastomosis with every reason for regurgitation of gastric juice, he has had complete healing of the esophagus.

The second case I think is more important. This was a woman 42 years of age with a long-standing history of dysphagia due to cardiospasm. She had a transthoracic lateral anastomosis done in 1949. She developed bleeding, due apparently to peptic esophagitis. Yet, on complete workup, she has a complete achlorhydria. The problem, I think, will have to be solved by methods other than those utilized at present, and I would like to call attention to the very stimulating article in a recent issue of *Surgery, Gynecology and Obstetrics* by Allison, of Leeds, England, in which there is described a new approach to the anatomy and physiology of the cardia and the esophageal diaphragmatic opening. I think possibly the answer will be a local resection with an anastomosis, or a lateral anastomosis, with reimplantation of the cardiophrenic ligaments so as to restore the pinchcock action of the two crura which was so well brought out by Mr. Allison in his article.

It is a curious observation that, in doing resections of the esophagus for carcinoma, one is never bothered by esophagitis or hemorrhage at a later date. Perhaps the vagal resection done at operation is a factor and perhaps in the operation for cardiospasm, vagotomy should be an added maneuver.

DR. E. K. JOHNSON, Brooklyn, N. Y.—We have seen a few children or infants with achalasia, or narrowing of the esophagus, at the pharyngo-esophageal junction. Perhaps the most interesting case was a young girl of 12 years, who was seen because of difficulty in swallowing. A tonsillectomy had been performed when she was 3 years old, after which she refused to take anything but liquids.

(Slide) This shows the pharynx outlined by barium which failed to enter the esophagus. She refused to take any more.

(Slide) The lateral view, at the pharyngo-esophageal junction, shows the narrowing.

(Slide) The postoperative esophagram reveals good filling. Using a cervical approach, the narrowed part of the esophagus was located. We found a thickened muscular band which had completely constricted the esophagus. Using a modified Heller procedure, the band was divided on the left side and a large catheter was passed without difficulty by the anesthetist.

(Slide) The diagram shows the site of the constricting band.

(Slide) We thought the band might be fibrous, but it was entirely muscular.

Her postoperative course has been very smooth and she eats her meals in normal fashion and has gained considerable weight. I thought this case was so similar to congenital hypertrophic pyloric stenosis and cardiospasm that it may prove of some interest here.

DR. M. E. DE BAKEY, Houston, Texas.—About ten or eleven years ago Dr. Ochsner and I reviewed this subject with particular reference to its surgical considerations. Among the various terms employed for the disease we preferred the term achalasia because we were inclined toward the pathogenic concept implied by it. It was pointed out at that time that the multifarious designations for the disease, as well as the various types of therapeutic procedures advocated for its treatment, reflected the state of confusion in our understanding of the disease. After listening to the discussions today I feel like Dr. Sweet, that this state of confusion has not been materially changed.

Critical evaluation of results of any operation proposed for this condition is highly important. At the time of our review and evaluation of the various procedures proposed, which are virtually the same as those presented today with the exception of the more radical procedure suggested by Dr. Wangensteen, we were impressed with the fact that most of them were enthusiastically reported to provide excellent results. This is particularly true for the so-called Heller operation which has again been enthusiastically advocated today. The fact that this operation was proposed by Gottstein approximately fifty years ago, first performed by Heller almost forty years ago, subsequently modified by a number of other surgeons, and abandoned and revived from time to time, strikes me as significant. Such considerations in the historical development of an operative procedure usually reflect dissatisfaction with its ultimate results as well as with its rationale. For these reasons it would seem desirable in the current revival of this operative procedure to provide additional facts for its rationale. The question which needs to be answered is why is it a better operation now?

The etiology of the disease remains unknown and, so far as I know, during the past decade little has been added to our knowledge of the condition to provide an adequate explanation of its pathogenesis. This is probably the main reason for the difficulties encountered in achieving an entirely satisfactory operative procedure. Our experience has led us to consider the Heyrovsky procedure with slight modifications to be the one of choice. I think the point that Dr. Sweet has made with regard to placement of the diaphragm in relation to the stomach is important, because it may help prevent regurgitation and the occurrence of esophagitis. Like others, we have observed the development of esophagitis and hemorrhage following operation, but in our hands it has occurred rather infrequently. It should be realized, however, that these complications also occur in some patients who have had no operative treatment.

The final point I would make is that, so far as etiology is concerned, I have serious doubt that this condition is related to hypertrophic pyloric stenosis or to mega-esophagus. This, too, is an idea which has been revived and abandoned from time to time, and to revive it again requires additional facts to support it.

DR. W. B. CRANDELL, White River Junction, Vermont.—About a year ago there appeared in the British Medical Journal an article by a group from Budapest on the effect of 100 c.c. of 1 per cent oral procaine on relaxing the pylorus and pylorospasm either primary or secondary to other organic disease. This was studied by roentgenogram and fluoroscopy with barium techniques and found quite successful in bringing about relaxation in a good percentage of nearly 1,500 cases that were studied. In July of last year we began a similar study and found it helpful in preparing patients who had pylorospasm associated with pyloric obstruction for operation. It was not helpful in those people who had organic stricture or carcinoma.

About two months ago we became interested in using it in esophageal disease and in cases with symptoms associated with hiatus hernia or duodenal ulcer. We did have recently one case with cardiospasm, and I would like to describe our experience with that. This was a 28-year-old man who was admitted with an eight months' history of intermittent vomiting after meals. He had had daily vomiting for two months prior to admission. He had epigastric and substernal pressure and obtained relief after vomiting. There was no relief from antacids, and he had lost 30 pounds during this period. Roentgenograms revealed cardiospasm.

(Slide) Esophagoscopy did not show any ulceration or marked esophagitis. There was slight but not much relief with belladonna and amphogel. A few days after admission he was started on oral procaine, 100 c.c. of 1 per cent solution a half hour before meals three times a day. At noon the first day he took his first second helping in several months. Two days later he had noticed marked improvement and was essentially asymptomatic. He gained some seven pounds in ten days. We tried to get some objective measurement of the effect of the oral procaine by having him fluoroscoped and x-rayed, using a stopwatch. The roentgenogram on the left shows what was found four minutes after a barium meal. The radiologist tells us that the level is at the level of the pulmonary artery. He was allowed to clear his esophagus and then was given 100 c.c. of 1 per cent procaine, followed in fifteen minutes by another swallow of barium which the radiologist tells us, five seconds later, is already down to this point, just above the diaphragm. Four minutes afterward, however, there was still some barium in the lower end of the esophagus.

Our experience with these few cases leads us to think that oral procaine might be useful in conjunction with antacid treatment for spasm of the lower end of the esophagus associated with ulcer, tumor, or cardiospasm in preparing patients for operation, particularly those who are malnourished.

DR. ARTHUR M. OLSEN, Rochester, Minn. (closing).—I should like to thank the discussors for their kind remarks and constructive criticism. In view of the fact that many papers concerning surgical management of cardiospasm have been published recently, we believed that attention should be called to the fact that the majority of patients can be treated satisfactorily by forceful dilatation of the cardia. As Dr. Tuttle* has emphasized, dilatations must be performed expertly. It is our opinion that anyone who is proficient in the techniques of bronchoscopy and esophagoscopy also should be capable of treating esophageal obstruction by the method of dilatation. Surely dilatation therapy should be tried in practically every case before operative intervention.

Dr. Sweet is certainly right when he states that the actual obstruction is located above the cardia in the lowermost segment of the esophagus. The term "cardiospasm" does not accurately describe the condition, but it has been used for so long that we find it difficult to change the terminology. There does appear to be a physiologic sphincter in the lower part of the esophagus which does not open readily. Although the word "achalasia" may come closer to describing the nature of the disease, it seems likely that "cardiospasm" will persist in medical literature for some time to come.

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*Dr. Tuttle's discussion was not returned.—Editor.

THORACOPLASTY COMBINED WITH RESECTION FOR PULMONARY TUBERCULOSIS[*]

William S. Conklin, M.D., John E. Tuhy, M.D. (by invitation), and
Jerome T. Grismer, M.D. (by invitation)
Portland, Ore.

Pulmonary resection has been accepted as an indispensable adjunct in the treatment of an increasing number of cases of pulmonary tuberculosis. It is extremely important therefore, to determine how the complications and unfavorable sequellae of this type of surgery may best be prevented and managed. Because of the unpredictable behavior of the disease, resections for tuberculosis have involved greater hazards than those for most other types of bronchopulmonary pathology. Specific complications include postoperative spreads, reactivation or progression of pre-existing lesions in nonresected lung, tuberculous bronchitis and ulceration in the stump of an amputated bronchus, and tuberculous empyema. Bronchopleural fistula is a greater hazard not only because tuberculous involvement of the bronchus may interfere with healing and predispose to perforation or rupture of the bronchial stump, but also because tuberculous foci may be traversed in dividing incomplete fissures or in separating the segments of a lobe. Antibiotics have markedly reduced, but have not eliminated, these complications of pulmonary resection for tuberculosis.

In all pulmonary resections it is important to preserve as much respiratory function as possible. When a lung, or part of a lung, has been removed the remaining lung tissue becomes distended or emphysematous, as its volume is increased to fill the relatively fixed volume of the thoracic cavity. Overdistended lungs are considered to function with impaired efficiency. It is also believed that residual tuberculous foci are more likely to manifest reactivation or progression as overdistention develops. Following pneumonectomy, space-filling substances (air, oil, plastic spheres, plastic sponge, etc.[3]) have been introduced into the pleural cavity to prevent such overdistention, and also to prevent excessive mediastinal displacement and the cardio-circulatory embarrassment which may result. But the use of space-filling materials in the pleural cavity following resection of only part of a lung does not seem practicable nor can it be expected to affect the incidence or management of fistula and empyema following any lung resection except, possibly, in an unfavorable manner.

When only a lower lobe or basal segments of a lower lobe are resected, space-filling does not present as great a problem. The hemidiaphragm tends to rise and obliterate the space. And this can be promoted readily, when necessary, by a phrenic nerve interruption, by pneumoperitoneum, or both.

From the Department of Surgery, Division of Thoracic Surgery, University of Oregon Medical School, and the Service of the Matson Memorial Hospital for Diseases of the Chest.

[*] Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

Many surgeons believe that a thoracoplasty should be performed routinely two or more weeks following most resections, particularly following removal of a tuberculous upper lobe or lung. The thoracoplasty, thus performed, tends to relieve lung distention and permits the mediastinum to return to a more normal position. It probably decreases the incidence of postoperative spread and exacerbation. Undoubtedly it helps to prevent the late development of bronchopleural fistula and empyema and aids in the control of these serious complications when they occur.

Some surgeons recommend that thoracoplasty be performed in advance of a contemplated resection. A few advocate performance of resection and thoracoplasty at the same operation. Whether performed earlier, concomitantly, or later, the thoracoplasty at present seems to offer the best solution to some of the problems connected with resection of a lung, or of an upper portion of a lung, particularly in patients with pulmonary tuberculosis. Granted the desirability of thoracoplasty for these patients, the authors propose to explore the advantages of the combined operation as borne out by their own experiences and those reported by others.

In May, 1948, we resected the left lung of a 39-year-old woman who had had a previous left thoracoplasty. Believing that the thoracoplasty should be extended, we decided to remove segments of three more ribs after the lung had been removed and before completion of the operation. Resection of these additional rib segments was well tolerated. This experience suggested to us that patients undergoing upper lobe lobectomy could properly have a partial thoracoplasty performed at the same time, without appreciably increasing the hazards of the surgery. When we next resected a tuberculous left upper lobe (July, 1948), we performed a five-rib thoracoplasty immediately. The additional surgery did not affect the patient adversely and the postoperative course was remarkably smooth.

We now plan most resections with the idea of a combined procedure in mind. Our resections are followed immediately by partial thoracoplasty when the advisability or need for thoracoplasty seems evident and when the patient's condition permits. We are convinced that definite advantages accrue.

The first combined pneumonectomy and thoracoplasty of which we have knowledge was the historic resection for carcinoma of the lung performed by Evarts Graham in 1933. At the time of pneumonectomy ribs three to ten were resected and later the first two ribs were removed in order to collapse a small empyema cavity. The empyema would doubtless have been a much more serious complication if the pleural dead space had not been largely obliterated at the time of the original operation. Dr. Graham performed thoracoplasty at the time of pneumonectomy in his next five or six cases, after which he discontinued this practice as a routine. However, he and Dr. Burford still frequently perform thoracoplasty in conjunction with pneumonectomy for tuberculosis in order to diminish the possibility of tuberculous empyema.[7]

The first publication devoted to the subject of combined resection and thoracoplasty was that of Iverson, Roland, and Skinner,[4] who recorded three cases of pneumonectomy for tuberculosis followed by immediate thoracoplasty. Seven additional cases were reported by two of these same authors at the 1949 meeting of the American Association for Thoracic Surgery.[5]

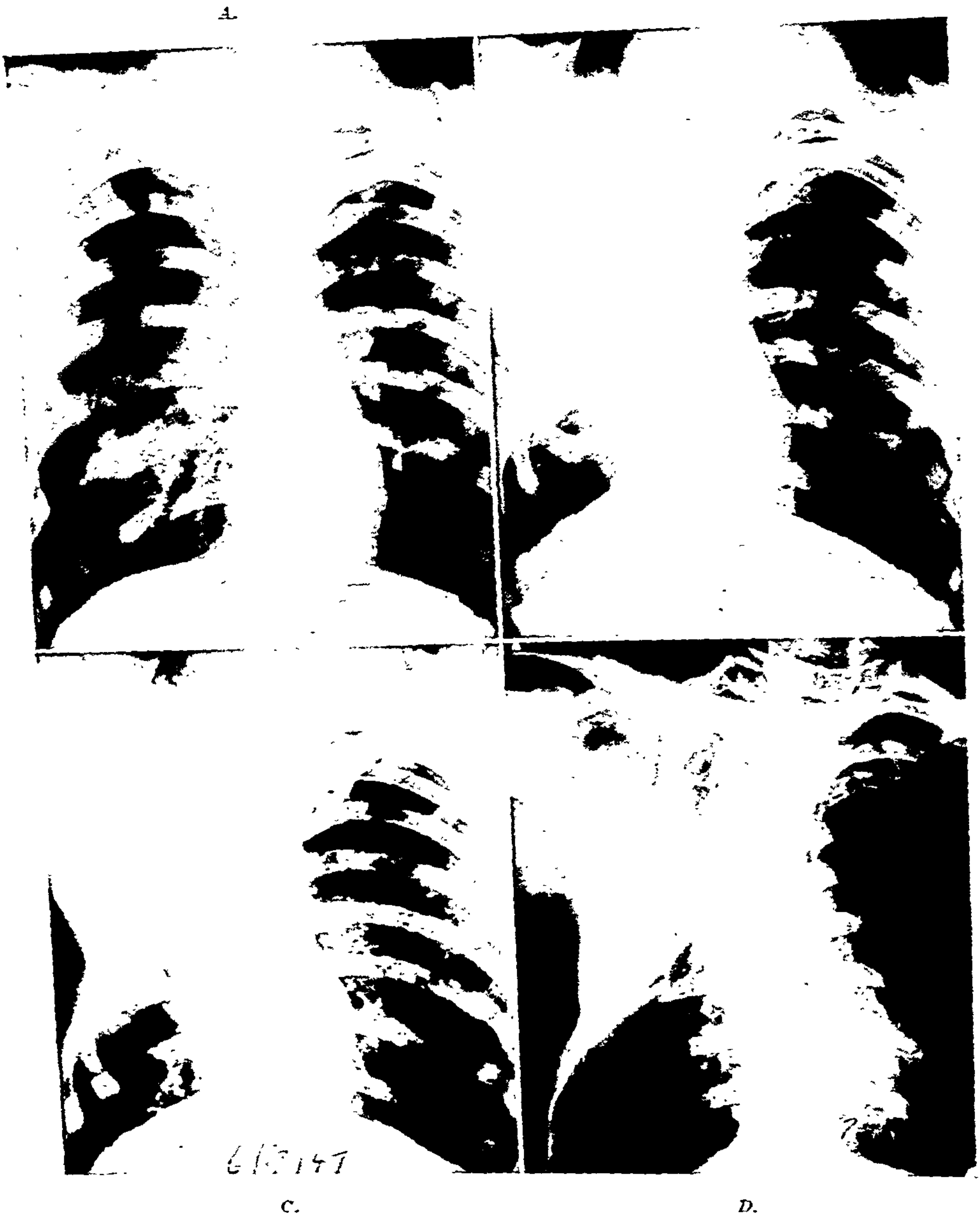

FIG. 1.—H. E. *A*, Inadequate pneumothorax with large cavity in atelectatic upper lobe. *B*, Upper lobe lobectomy, followed by early thoracoplasty in two stages. First rib not resected. *C*, Bronchogenic tuberculous dissemination. *D*, Bucky roentgenograph shows evidence of empyema and bronchopleural fistula (late development) which were, apparently, the source of bronchogenic spread. The empyema and fistula were eventually controlled by drainage and revision thoracoplasty, including the first rib. The tuberculous spread responded to antibiotic therapy.

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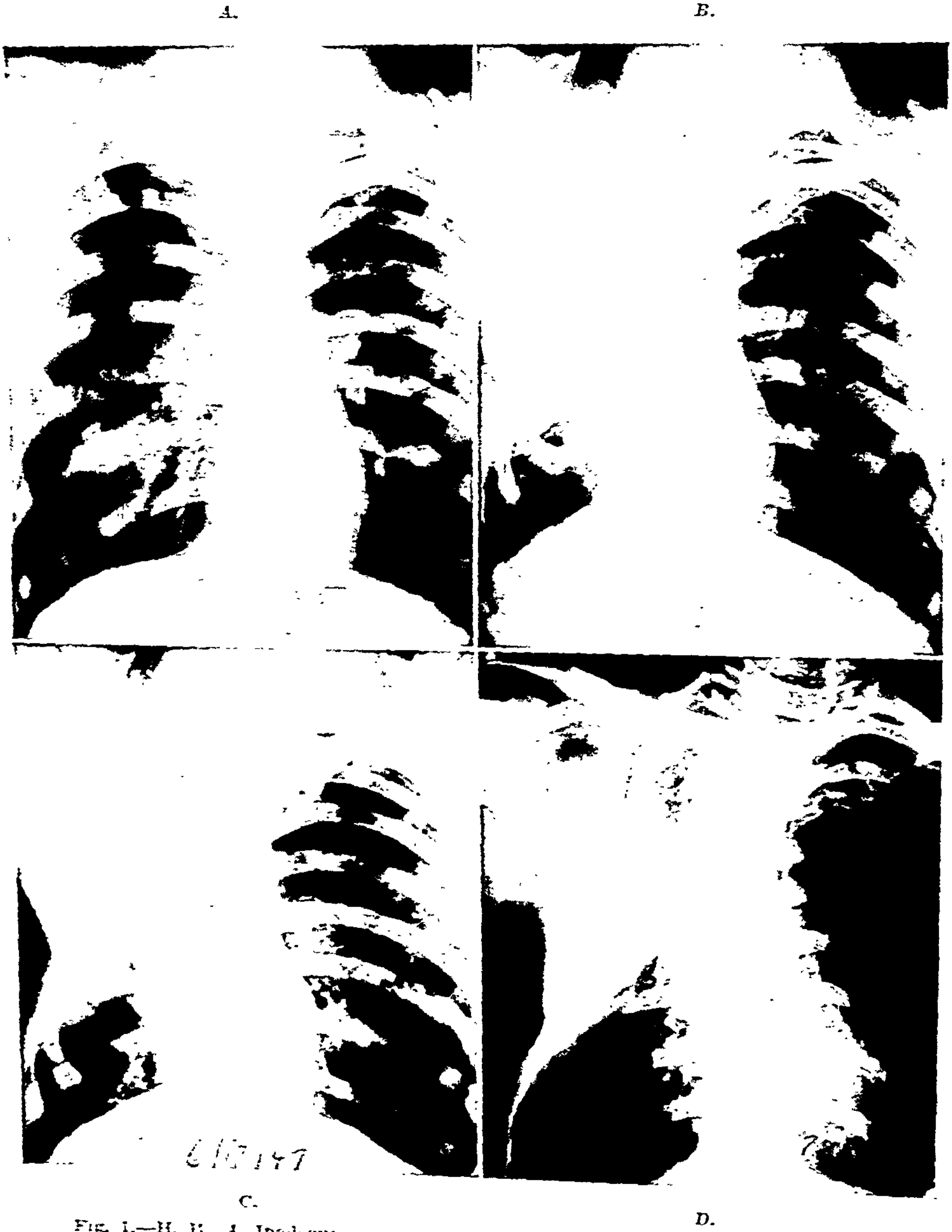

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Iverson and Skinner had been impressed by the relatively benign postoperative course following pulmonary resection when a thoracoplasty had previously been performed, despite the increased technical difficulties of postthoracoplasty resection. They had also been disturbed by the fact that some patients would refuse a recommended thoracoplasty while convalescing from a pneumonectomy. In the light of their experiences with the combined operation they concluded that combining thoracoplasty with pneumonectomy is advantageous when overdistention of the contralateral lung is particularly undesirable, as in the presence of emphysema, and when postoperative pleural complications are anticipated. They emphasized the points that when thoracoplasty has been performed with pneumonectomy the danger of postoperative fistula and empyema is decreased, and the treatment of these complications is easier if they occur. They also concluded that the combined procedure resulted in less disturbance in respiratory physiology than one is likely to encounter following pneumonectomy alone. This they attributed to the weight of the scapula, combined with use of a pressure dressing over the decostalized area, rendering the mediastinum relatively stable and the cough mechanism more effective. Weight was added to their arguments for performing thoracoplasty at the time of pneumonectomy, rather than later, by quoting from Minor[6]: "When a thoracoplasty is performed over a closed pleural space containing air, or fluid, or both, a rapid increase in intrapleural pressure results. Highly positive pressure upon the heart and mediastinum produces a form of shock which may be rapidly fatal." They also quoted Bailey[1]: "Since two of the five deaths were subsequent to elective thoracoplasties performed merely to obliterate the pleural space, it can well be imagined that we are on the lookout for a method of avoiding this secondary operation."

Our material and technique have differed from those of Iverson and Skinner. In the first place, while they used the combined procedure only where total pneumonectomy was performed, we have used it more often with upper lung resections than with pneumonectomy. The above authors routinely left the first rib (or the first and second ribs) intact, as advocated by Overholt in postresection thoracoplasties. However, we have preferred to remove the uppermost ribs, believing that this helps to obliterate the pleural dead space more rapidly and prevents significant displacement of the trachea and superior mediastinum (Figs. 1 and 2). Iverson and Skinner have routinely drained the pleural space in patients who have had combined pneumonectomy and thoracoplasty, but we have not done so. There seems to be more rationale for drainage following the combined operation than after pneumonectomy alone, since the thoracoplasty, supported by a pressure dressing, will prevent excessive mediastinal shift, and drainage will promote obliteration of the pleural cavity. We routinely use one or two drainage tubes when lobectomy or segmental resection has been combined with thoracoplasty.

A paper on upper lobe lobectomy and concomitant thoracoplasty, by Samson, Dugan, and Harper, was published in December, 1950.[8] The authors report seventeen such operations with one death and no postoperative complications. They also report nine pneumonectomies and concomitant thoracoplasties with no complications or deaths. While our technique differs somewhat from theirs, our operative material and results have been similar and we seem to be in complete agreement as to the advantages of the combined procedure. The usual indications for resection were present in most of the patients who had concomitant thoracoplasty and resection in our series, though some of the resections may be considered "elective," in that other surgeons might have recommended thoracoplasty alone.

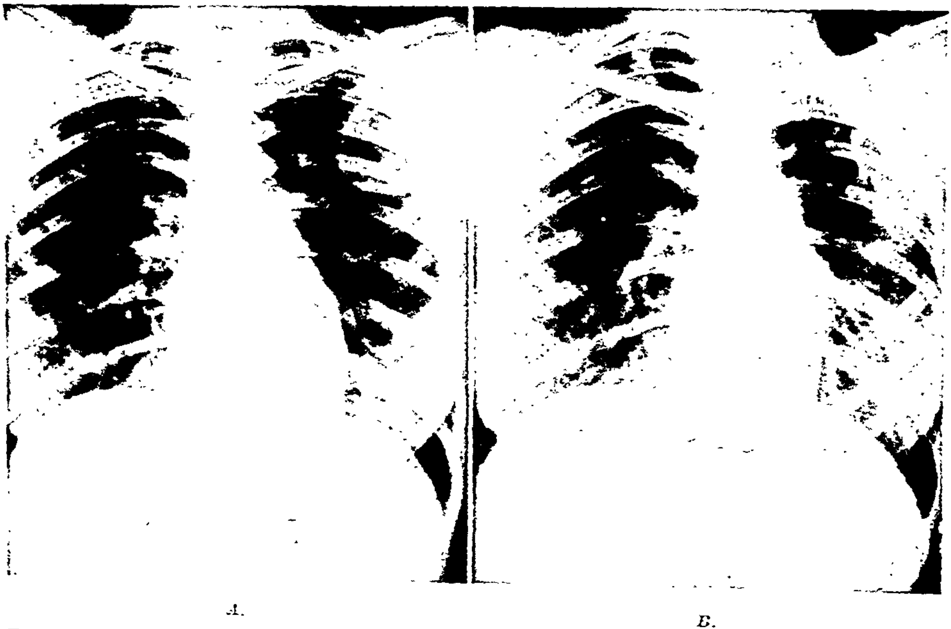


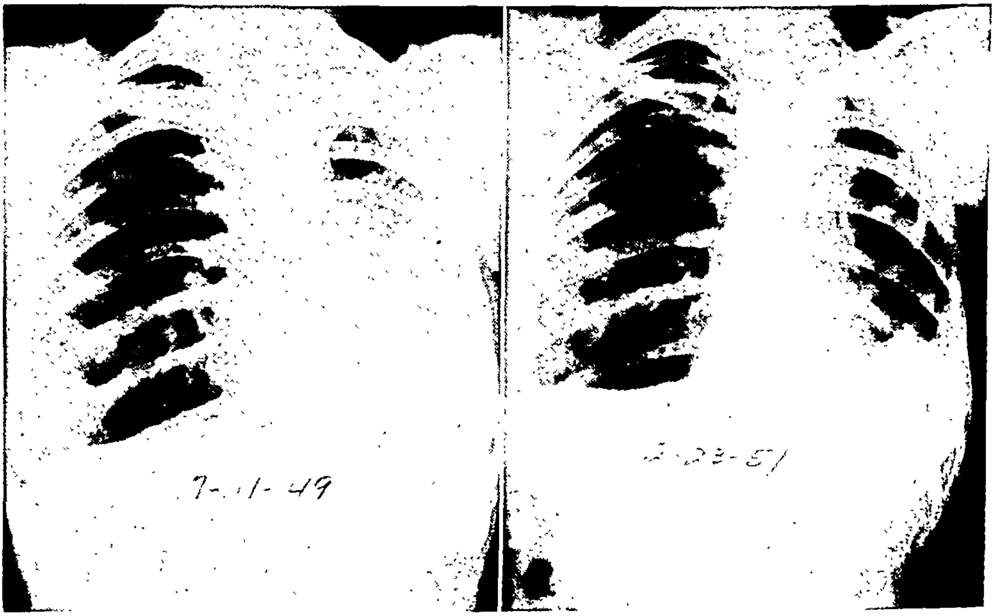
Fig. 2.—H. K., A, Admission roentgenograph. "Tension" cavity in left upper lobe. B, Following resection of left upper lobe with concomitant four-rib thoracoplasty.

TECHNIQUE

Standard methods of pre- and postoperative management were used. The Overholt face-down position was employed in only one operation. The exposure for resection of the upper ribs was found to be poorer than in the lateral decubitus position, which the authors prefer. Anesthesia was induced with Pentothal-curare solution injected intravenously, and continued with cyclopropane, nitrous oxide, ether, and oxygen, administered by endotracheal tube.

In our earlier cases the thoracotomy incision was made through the periosteal bed of the fifth or sixth rib. In upper lobe lobectomies it soon became evident that extensive resection of these ribs was generally not required for a thoracoplasty designed merely to obliterate the pleural dead space. We then found that, working through the same skin incision, we could limit resection of ribs to those segments directly overlying the portion of the lung to be removed.

Our revised technique involves the removal of a short posterior segment of the fourth rib, the pleural cavity being entered through its periosteal bed. The incision is extended along the fourth intercostal space, after dividing the fourth intercostal neurovascular bundle between suture ligatures. The fifth rib and interspace are used when a five rib thoracoplasty is contemplated. The exposure thus obtained is generally adequate for any type of resection. It can be increased by resecting at once that portion of the third (or fourth) rib, usually its posterior third, which would be resected for the proposed thoracoplasty. The trapezius and rhomboid muscles are divided either to a limited extent, or not at all.



A. *B.*

Fig. 3.—C. H., *A*, Patient has had left artificial pneumothorax, pleural exudate, and obliterative pleuritis. Sputum, positive. *B*, Eighteen months following resection of contracted left upper lobe, decortication of left lower lobe, and concomitant thoracoplasty. Patient clinically and subjectively well for the first time in ten years. Sputum, negative.

After the significantly involved lung segments have been resected, adhesions to the remainder of the lung are separated and decortication is performed if indicated (Figs. 3, 6, and 10). The patient's condition is then evaluated and it has almost always seemed safe and proper to complete the thoracoplasty by resecting portions of the ribs remaining above, including the first two ribs forward to, or almost to, the cartilages.

With the pleural cavity open, following the pulmonary resection, we feel that we can determine more accurately how much of each rib should be removed in order to achieve the desired amount of chest wall collapse. The extent of the thoracoplasty can be adapted to the remaining portion of the lung, without collapsing conserved lung unless one specifically wishes to do so because of residual tuberculous foci. We wish to emphasize that the open thorax provides adequate exposure for completion of the upper phase thoracoplasty through an incision that does not involve extensive division of the interscapular shoulder muscles. The posterior rib stumps are purposely left long. The transverse processes and erector spinae muscles are not disturbed. This modified type of thoracoplasty seems to provide the desired amount of collapse with minimal postoperative scoliosis, chest deformity and impairment in function of the shoulder girdle.

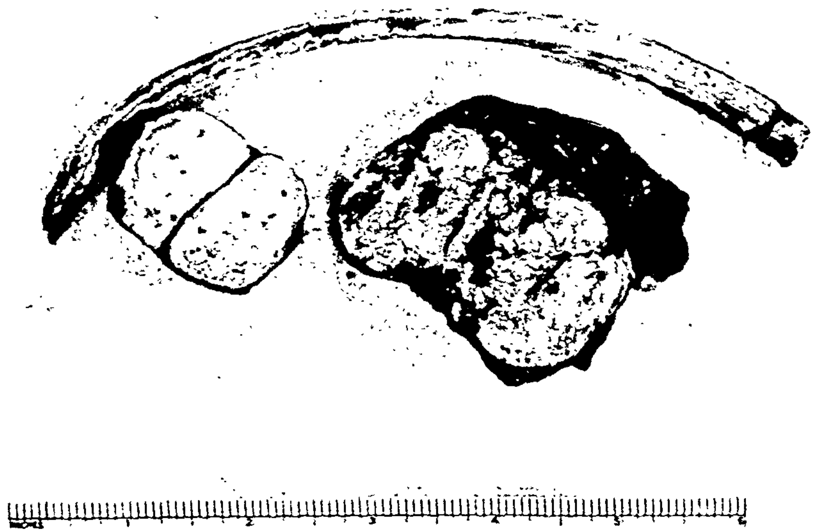


Fig. 4.—E. L., *A*, Admission roentgenograph. *B*, Body section radiograms showing a tuberculous granuloma with central necrosis in the right upper lobe (11 cm. cut) and a contracted superior segment of the right lower lobe with cavities (4 cm. cut). *C*, Following bilateral artificial pneumothorax and antibiotic therapy resection of the superior segment of the right lower lobe and of the "tuberculoma" in the right upper lobe (local resection) has been performed. Remainder of the lung at surgery showed no residual gross involvement and, when expanded, its volume was such as to make thoracoplasty appear unnecessary. Left pneumothorax has been maintained and sputum is negative. Resection of "tuberculoma" in left lung may be advisable.

In one of our patients who had mixed infection empyema, bronchopleural fistula and tuberculous bronchiectasis, eight ribs were removed at the time of total pleurectomy and pneumonectomy. Ordinarily, however, a four or five rib thoracoplasty is performed and, if additional collapse is deemed advisable, a second stage lower phase thoracoplasty is carried out two or three weeks later.

CLINICAL MATERIAL

We have not chosen patients for concomitant resection-thoracoplasty by any fixed protocol. Usually the preoperative plan, tentatively recommended at staff conference, has been carried out without alteration. In some patients thoracoplasty is considered unnecessary because the nonresected portion of the lung fills the pleural cavity sufficiently to obviate excessive overdistention and mediastinal displacement. This is often true following resection of

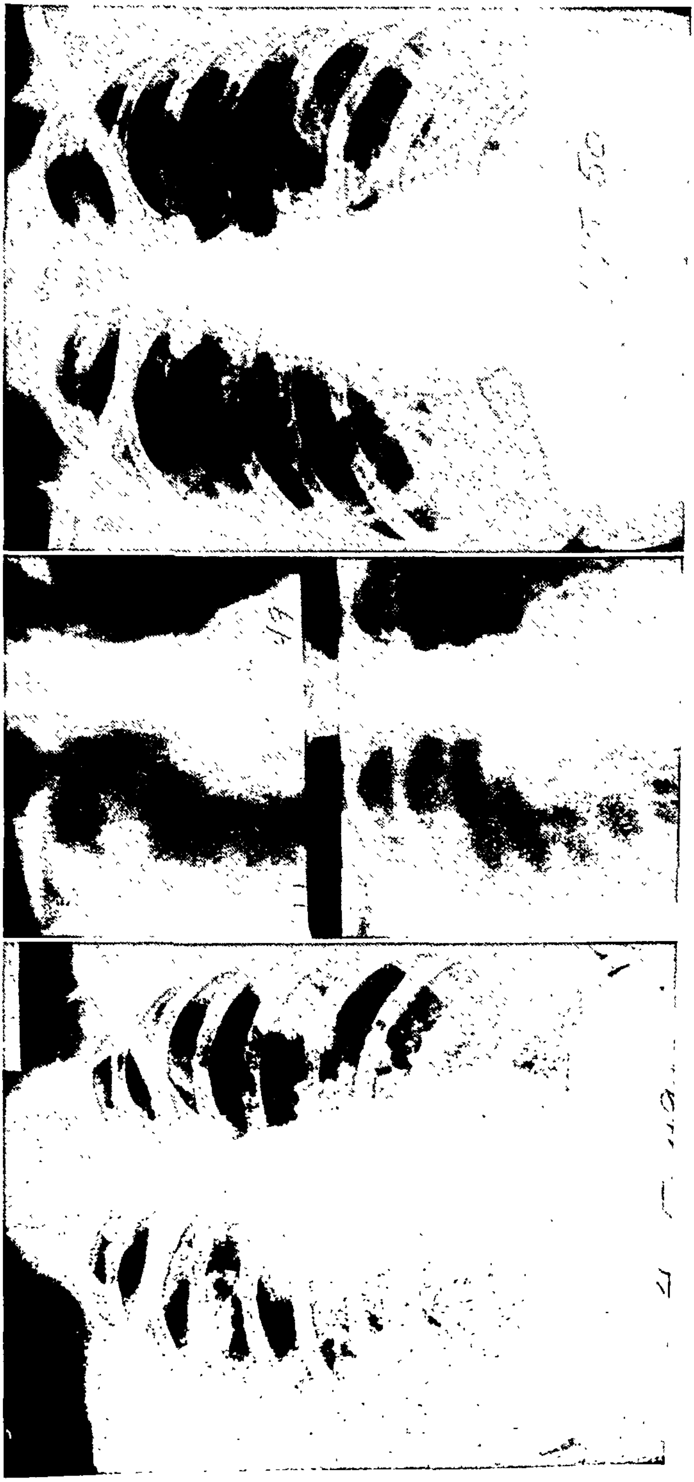


Fig. 4 *D.*—E. L. Surgical specimens. Today, resection of the long rib segment would have been avoided.

a markedly contracted lobe or following segmental and local resections, as for "tuberculoma" (Fig. 4). The combined procedure may sometimes be chosen at the time of surgery, because expansion of the remainder of the lung is poorer than anticipated (Fig. 10) or because it is felt that thoracoplasty provides greater protection for tuberculous lesions which are grossly evident, but not resected. In a few patients we have contemplated the combined procedure, but postponed the thoracoplasty because, following resection, the patient's condition did not warrant the additional operative trauma and blood loss.

We have performed thirty-eight combined resection-thoracoplasties between May, 1948, and April, 1951. There were twenty-eight women and ten men, all white, ages ranging from fifteen years to fifty-seven years. Most of the patients had far-advanced bilateral disease and had had one or more previous hospitalizations for tuberculosis. About two-thirds had had previous collapse therapy. One patient in the lobectomy group and two in the pneumonectomy group had had a previous apical thoracoplasty which was extended at the time of resection. One patient, listed with the upper lobe resections, had previously had the homolateral middle and lower lobes removed.

Of the thirty-eight patients, six were subjected to pneumonectomy. In twenty-nine an upper lobe was removed and in twelve segmental resection was performed alone or in conjunction with lobectomy.

COMPLICATIONS

The complications are listed in Table II.

Bronchopleural fistula occurred twice. In the first of these the fistula became apparent on the thirteenth postoperative day. This was controlled by

TABLE I

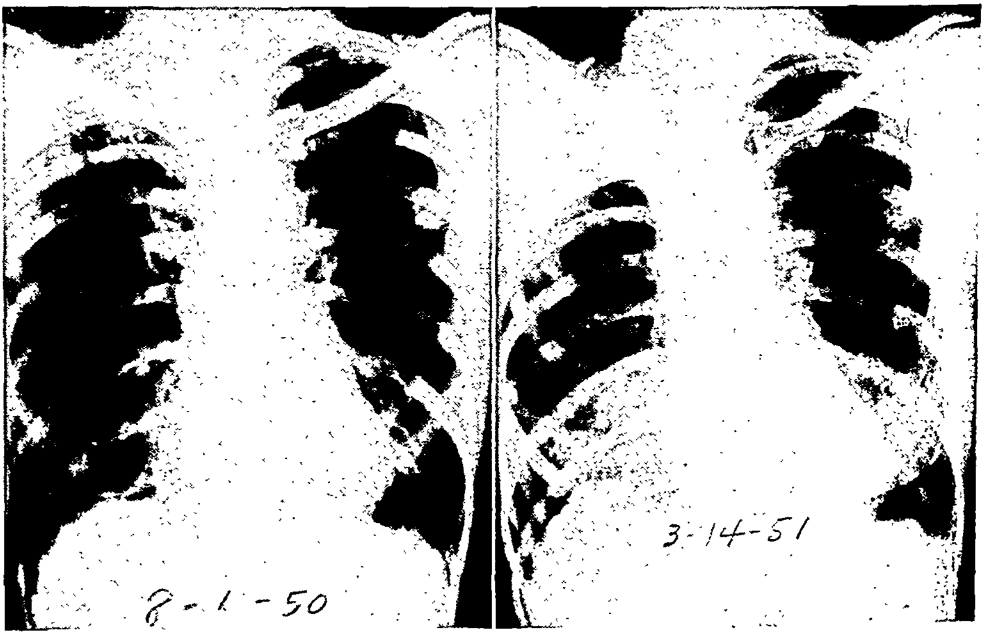
	NO. OF PATIENTS
Upper lobe *only*	18
Upper lobe, superior segment of lower lobe	8
Same, plus *right middle lobe*	1
Right upper and right middle lobes	2
Segment of upper, only	2
Superior segment of lower, only (Fig. 10)	1
	—
	32
Pneumonectomy (left, 5; right, 1)	6
	—
	38

TABLE II. POSTOPERATIVE COMPLICATIONS

	LOBECTOMY	PNEUMONECTOMY
Bronchopleural fistula	2	
Early contralateral spread	1	
Atelectasis	1	
Recurrent nerve palsy		
Pulmonary edema, atelectasis, right heart failure	1 (died)	1
	—	—
	5	1

introduction of an intercostal tube which was left in place for five days. Clinically there had been no evidence of empyema and the patient's convalescence was otherwise uneventful (Fig. 5).

The second bronchopleural fistula occurred in a recent case and this was further complicated by an empyema. The combined operation involved resection of the apical segment of the right upper lobe and segments of only the first three ribs, a slight departure from our usual technique. Since the original surgery, decortication for the empyema and resection of two more ribs has been performed in two stages. The patient is now convalescing satisfactorily.

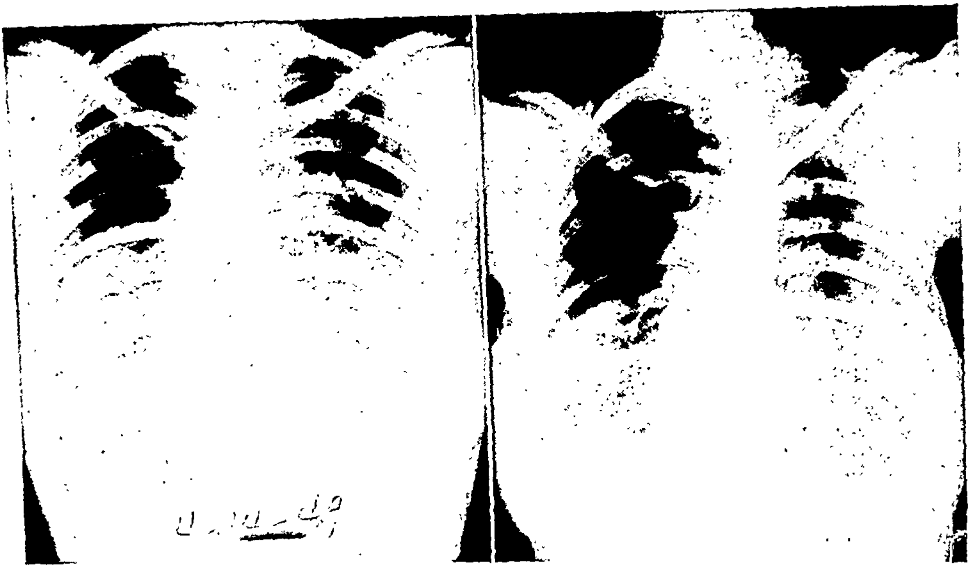


A. *B.*

Fig. 5.—E. F., *A*, Contracted upper lobe containing thick-walled cavity. *B*, Following resection of right upper lobe and concomitant thoracoplasty. Contralateral disease now appears stable. Sputum, negative.

One patient had an early postoperative spread, or exacerbation, in the contralateral upper lobe where pre-existing tuberculous lesions had been considered stable. This complication responded promptly to antibiotic therapy and the patient is clinically well more than one year following her discharge from the hospital. One patient had a temporary atelectasis following surgery which responded to aspiration of the tracheobronchial tree with no further difficulties. In a patient subjected to total pleurectomy and pneumonectomy for chronic mixed infection empyema with bronchopleural fistula and tuberculous bronchiectasis, present for many years, the left recurrent laryngeal nerve inadvertently was injured or severed. Left recurrent laryngeal nerve palsy has persisted.

The one death occurred in a 36-year-old white man, following resection of the right upper and middle lobes and a five-rib thoracoplasty for far-advanced cavitary disease. His condition was satisfactory until the fourth postoperative day when he developed abdominal distention and progressively increasing dyspnea. These did not respond to the usual management and he died twelve hours later. An autopsy revealed "acute massive edema of the left lung, bilateral pulmonary tuberculosis, purulent tracheobronchitis, atelectasis of the right lower lobe, dilatation of the right heart and paralytic ileus." It is believed that this death might have been avoided had his airway been cleared and his ileus treated more promptly.



A. *B.*

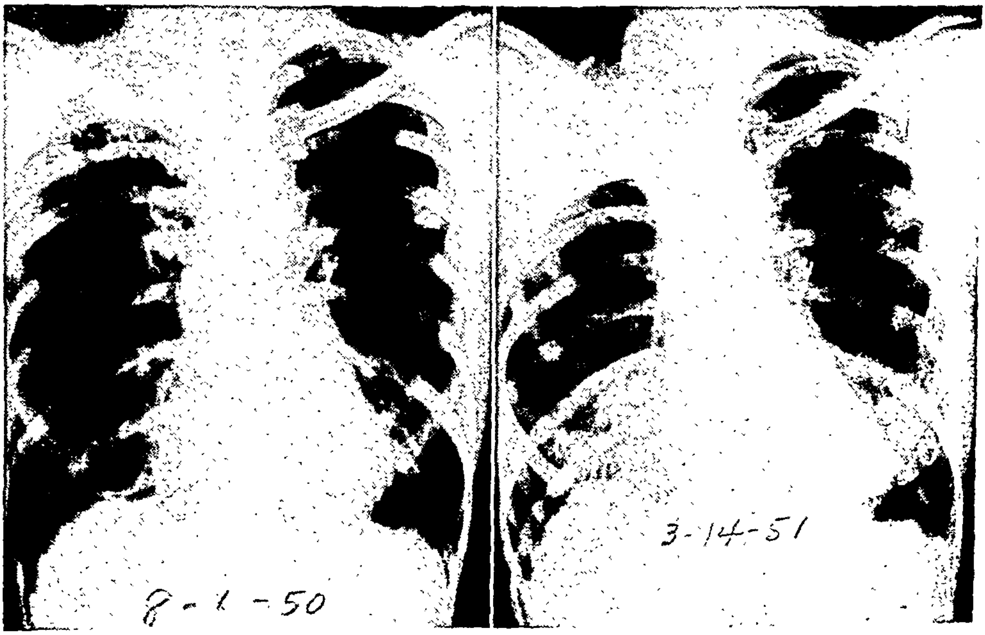
Fig. 6.—L. C., *A*, Prolonged bilateral artificial pneumothorax has been complicated by tuberculous exudate on both sides. Sputum, positive. *B*, Following resection of apical-posterior segment of left upper lobe, decortication of the remainder of the lung, and concomitant four-rib thoracoplasty.

RESULTS

The thirty-eight cases presented are too few for statistical analysis or for comparison with a series of similar cases subjected to resection alone or to resection with previous or subsequent thoracoplasty. The follow-up has not been long enough to draw conclusions about the long-term results. However, one can obtain an idea of the early postoperative morbidity from the data at hand.

There is only one patient who has a positive sputum and this is attributed to pre-existing contralateral disease. She is not hospitalized at present, but we consider her a candidate for concomitant right decortication, segmental resection and thoracoplasty, corresponding to the operation which she had performed two years ago on the left (Fig. 6). One patient is dead (see above). If one were to include the nine who are still hospitalized (and in all of them introduction of an intercostal tube which was left in place for five days. Clinically there had been no evidence of empyema and the patient's convalescence was otherwise uneventful (Fig. 5).

The second bronchopleural fistula occurred in a recent case and this was further complicated by an empyema. The combined operation involved resection of the apical segment of the right upper lobe and segments of only the first three ribs, a slight departure from our usual technique. Since the original surgery, decortication for the empyema and resection of two more ribs has been performed in two stages. The patient is now convalescing satisfactorily.



A. *B.*

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One patient had an early postoperative spread, or exacerbation, in the contralateral upper lobe where pre-existing tuberculous lesions had been considered stable. This complication responded promptly to antibiotic therapy and the patient is clinically well more than one year following her discharge from the hospital. One patient had a temporary atelectasis following surgery which responded to aspiration of the tracheobronchial tree with no further difficulties. In a patient subjected to total pleurectomy and pneumonectomy for chronic mixed infection empyema with bronchopleural fistula and tuberculous bronchiectasis, present for many years, the left recurrent laryngeal nerve inadvertently was injured or severed. Left recurrent laryngeal nerve palsy has persisted.

mary thoracoplasty would have had to be considerably more extensive than that combined with resection, and it would generally have involved two or more staged operations. We are of the opinion that in each instance the com-

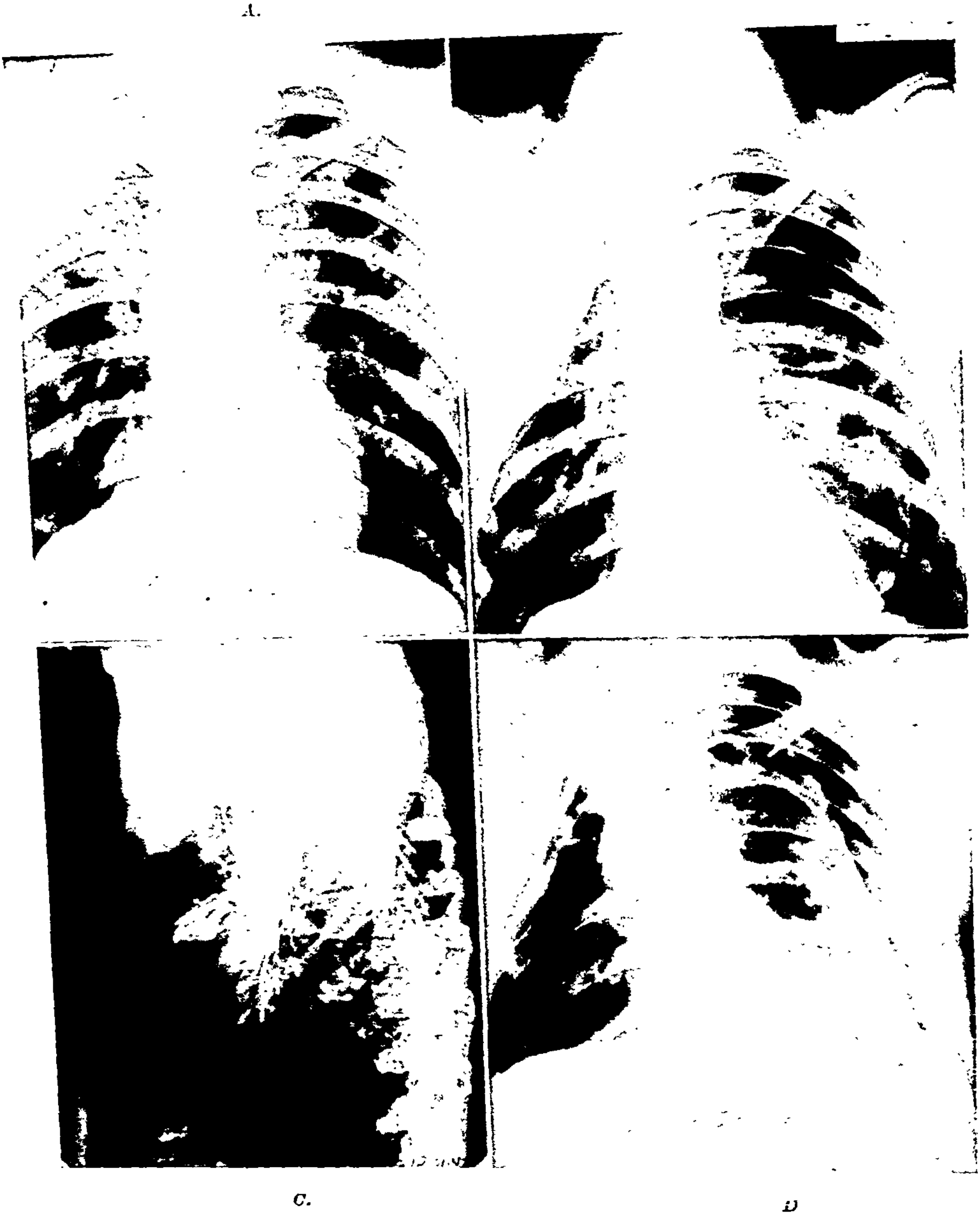

Fig. 7.—E. B., *A*, Admission roentgenograph. *B*, Since primary thoracoplasty on the right did not convert sputum, a left artificial pneumothorax was induced. Sputum still positive. *C*, Tuberculous bronchiectasis demonstrated in right upper lobe by bronchography. *D*, Sputum converted following resection of right upper lobe.

Comment: Recent experiences suggest that this patient would have had markedly decreased morbidity and far greater conservation of function had combined resection-thoracoplasty, supported by antibiotic therapy, been available to him. Compare with Fig. 5.

TABLE III. RESULTS

	ALIVE SPUTUM NEG.	ALIVE SPUTUM POS.	STILL IN HOSPITAL	DEAD
Lobectomy	30	1	6	1
Pneumonectomy	6	–	3	–
	36	1	9	1

Follow-up: More than two years	8
Between one and two years	10
Less than one year	19
Dead	1
	38

the prognosis is considered good) we find that thirty-six or 95 per cent have negative sputum and are candidates for permanent arrest of their disease.

We are impressed that these results, admittedly tentative, compare favorably with the early results of thoracoplasty alone or with those for any other type of therapy for tuberculosis. In evaluating this statement it should be remembered that most of the patients in this series had previously been subjected to other types of therapy without success and that most of them had a type of lesion for which resection is considered mandatory. Since thoracoplasty is considered advisable as an adjunct to resection for pulmonary tuberculosis, and since it does not appear to increase the hazard when, in selected cases, it is performed at the same time rather than separately, there seems to be ample justification for the combined operation.

DISCUSSION

We do not attempt, in this paper, to discuss the indications for pulmonary resection in tuberculosis. It is pertinent, however, that the authors try to avoid sacrificing an entire lung simply because the tuberculous pathology is found, at operation, to be more widespread than anticipated from preoperative roentgenographic interpretations. We prefer to abandon a resection in favor of some other type of therapy if, at thoracotomy, we find that a more extensive resection than was considered to be well within the patient's tolerance would be necessary. In general, we try to limit the resection to those portions of a lung that are completely destroyed or that contain a type of lesion notoriously resistant to medical and collapse therapy. It is our impression that remaining lesions in the same and contralateral lung will actually respond better to rest and antibiotics after the major pathology, such as caseating lesions and bronchiectasis, has been removed. This is perhaps especially true when concomitant thoracoplasty prevents distention of the remainder of the lung (Fig. 5).

It may be argued that in several instances where we have performed a combined resection-thoracoplasty it would have been preferable to perform the thoracoplasty first. It is possible that in some of these the thoracoplasty alone would have served to control the disease, but preoperative studies always made us think that such a result was quite unlikely. Certainly a primary thoracoplasty would have had to be considerably more extensive than that combined with resection, and it would generally have involved two or more staged operations. We are of the opinion that in each instance the com-

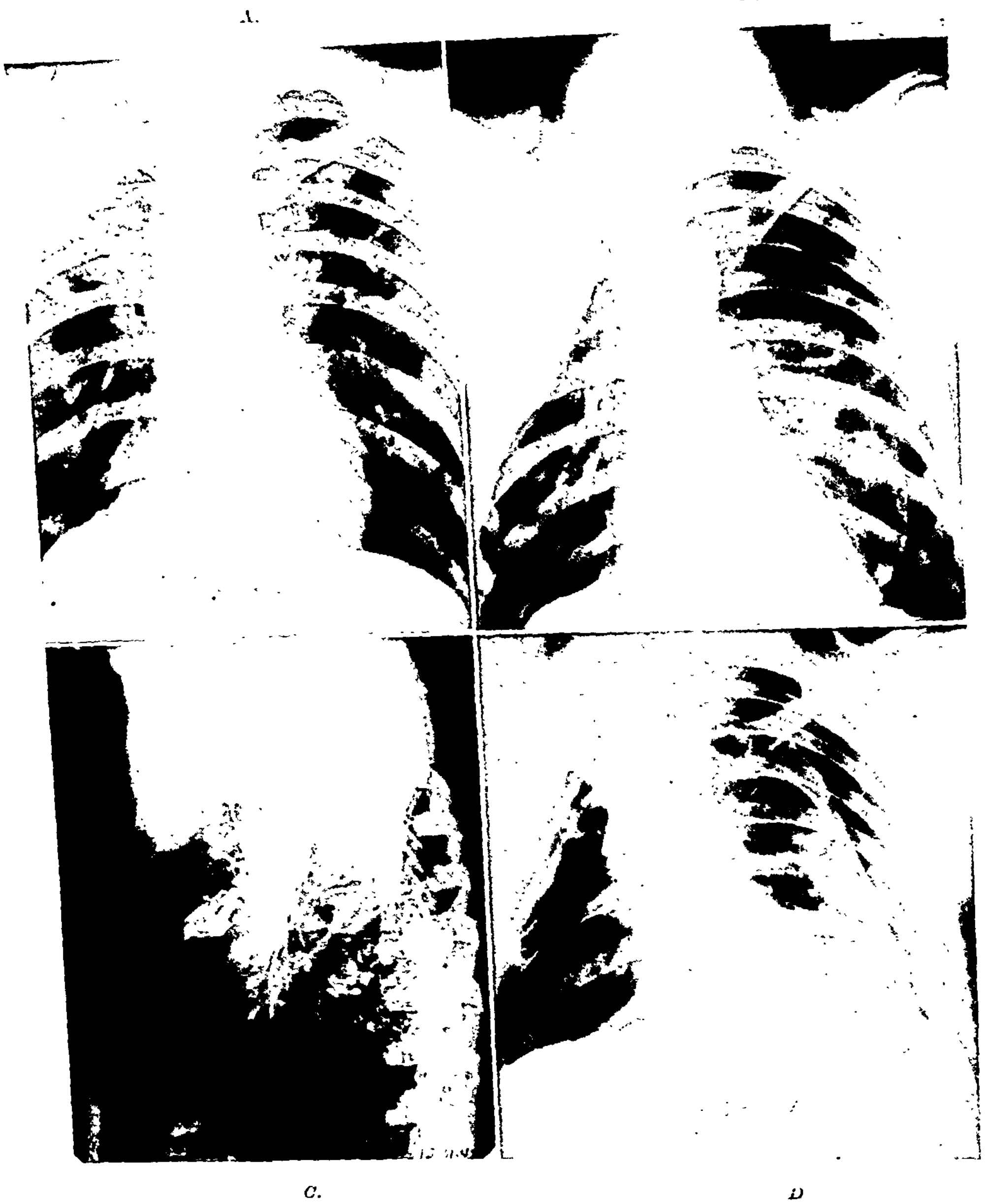

Fig. 7.—E. B., *A*, Admission roentgenograph. *B*, Since primary thoracoplasty on the right did not convert sputum, a left artificial pneumothorax was induced. Sputum, still positive. *C*, Tuberculous bronchiectasis demonstrated in right upper lobe by bronchography. *D*, Sputum converted following resection of right upper lobe.

Comment: Recent experiences suggest that this patient would have had markedly decreased morbidity and far greater conservation of function had combined resection-thoracoplasty, supported by antibiotic therapy, been available to him. Compare with Fig. 5.

bined operation has left the patient with more respiratory reserve, more shoulder function and less deformity than a standard thoracoplasty would have entailed.* *We also are inclined to believe that the combined operation* might have decreased the morbidity and eliminated the need for extensive bi-

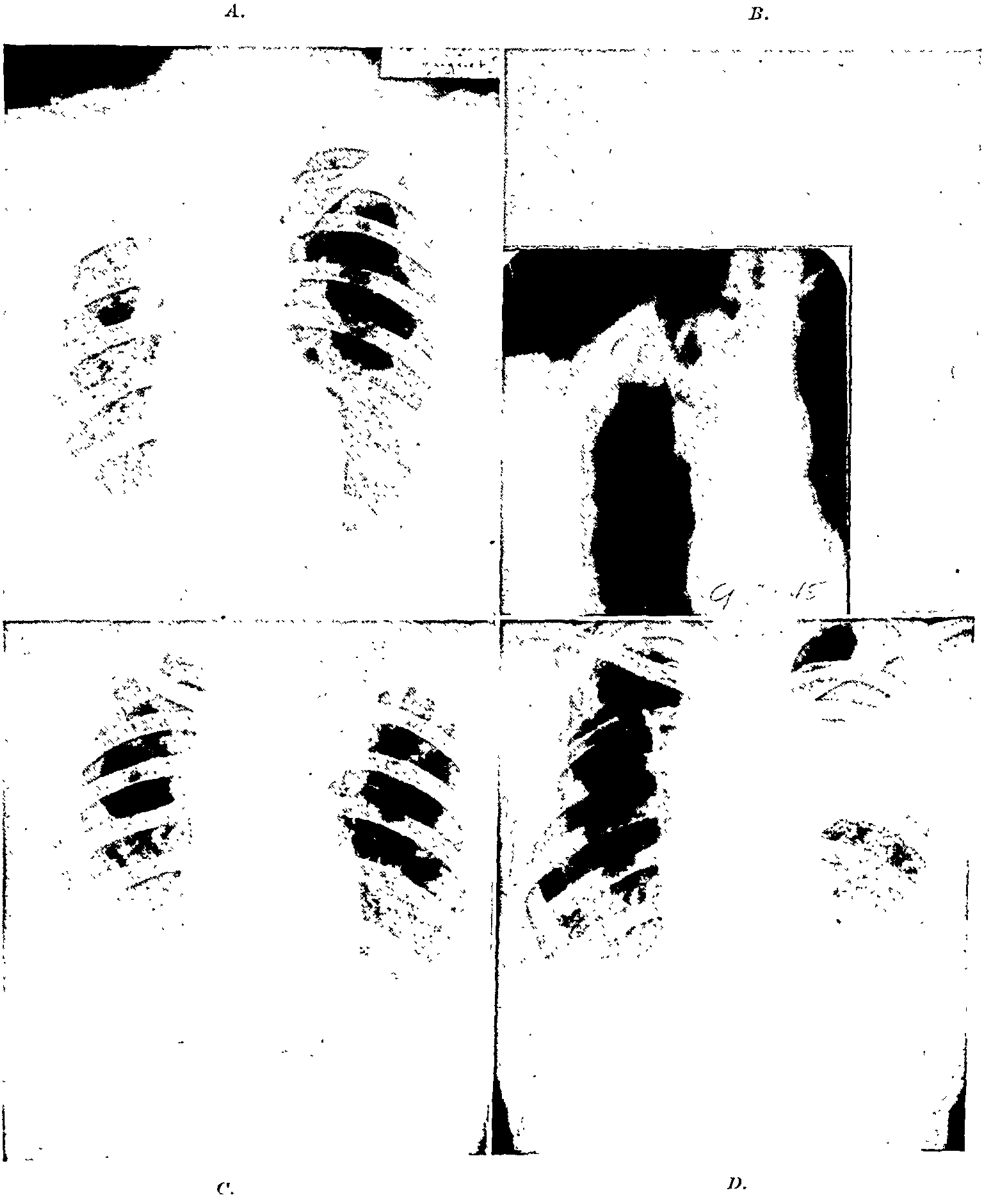

Fig. 8.—M. Y., *A*, Admission roentgenograph. *B*, Body section radiogram showing contracted upper lobe, bronchiectasis, stenosis of right upper lobe bronchus. *C*, Following resection of right upper lobe. Postoperative spread to left upper lobe. *D*, Following left extrapleural pneumothorax.

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*Samson and co-authors[5] state: "While facilities are lacking to substantiate this belief, it is the authors' impression that there is less reduction in the vital capacity following lobectomy-thoracoplasty than with a thoracoplasty which would have been sufficient to control the disease."

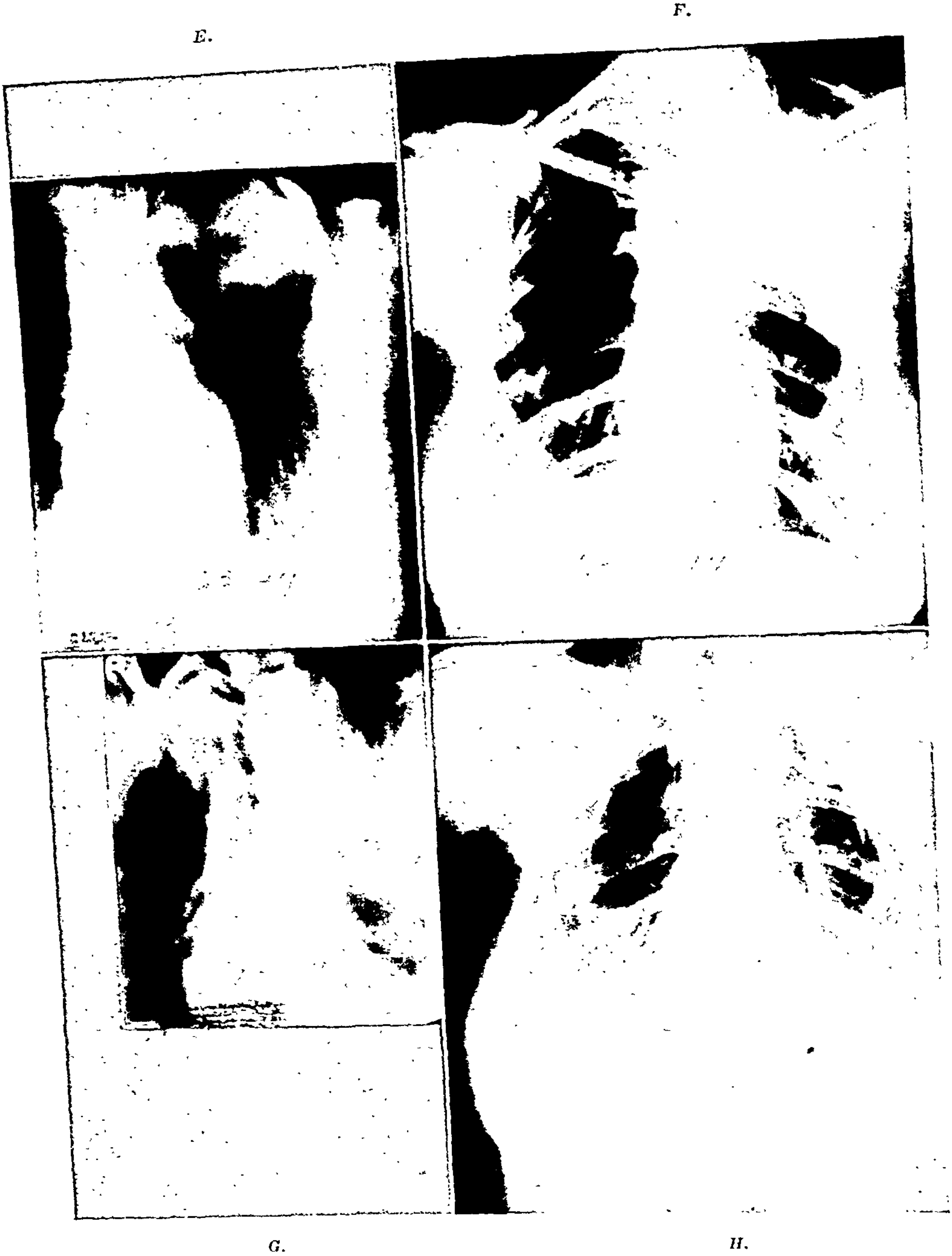


Fig. 8.—*E*, Sputum, still positive. Body section radiogram shows small residual cavity under extrapleural pneumothorax. *F*, Following left thoracoplasty. *G*, Oblique body section radiogram shows cavity in superior division of right lower lobe. *H*, Over three years following right thoracoplasty. Patient has arrested tuberculosis.

Comment: It is believed that this patient's tuberculosis might well have been promptly controlled today, by combined resection-thoracoplasty and antibiotic therapy without having to be subjected to the extensive collapse procedures illustrated. Note the overdistention of the right lower lobe following lobectomy, only partially relieved by subsequent thoracoplasty.

lateral collapse therapy in some of our earlier cases who had primary thoracoplasty, or who had resection without concomitant or early thoracoplasty. Examples are illustrated (Figs. 7 and 8).

A thoracoplasty performed in anticipation of a later resection subjects the patient to an additional major operative procedure, which we believe should be avoided. Following a thoracoplasty resection becomes technically more difficult and it is harder to dissect and free adhesions in the costovertebral gutter without tearing into infected lung tissue or a cavity. We also feel that, with the protection provided by antibiotics, significant postoperative spreads may, in years to come, be shown to occur less frequently following resection than following thoracoplasty, because the chief source of bacilliferous sputum is not merely collapsed, but removed.

An advantage in performing thoracoplasty in conjunction with lobectomy rather than later, suggested by Himmelstein, Berry, and Read,[3] is that the remainder of the lung becomes adjusted to the residual pleural space before its motility is limited by pleural adhesions. If the thoracoplasty is delayed and there has been an opportunity for adhesions to develop, the thoracoplasty will serve mostly to collapse the upper portion of the remaining lung tissue, leaving the rest of it overdistended (Fig. 8).

On the basis of our limited experience with the procedure we have tentatively adduced the following other advantages of the combined operation over resection and thoracoplasty performed separately:

1. The thoracoplasty need not be as extensive or radical as when it is performed before or after a subtotal pulmonary resection. Hence, there is less deformity, less disturbance to the function of the shoulder girdle, and probably less disturbance to respiratory function. If necessary, the thoracoplasty can later be revised and extended to control lesions in the nonresected portion of the lung though this has not as yet been necessary in any of our cases.

2. The extent of the thoracoplasty can be conformed rather accurately to the size of the pleural dead space before overdistention and adherence of the remainder of the lung have occurred.

3. It is probably safer, with the combined operation, to conserve portions of the lung containing lesions for which resection ordinarily would not be considered.

4. The combined procedure allows better exposure of the uppermost ribs through an incision which is shorter than that generally used for thoracoplasty alone. There is less damage to the muscles of the shoulder girdle and postoperative deformity (scoliosis) is minimized by preserving the muscles of the spine.

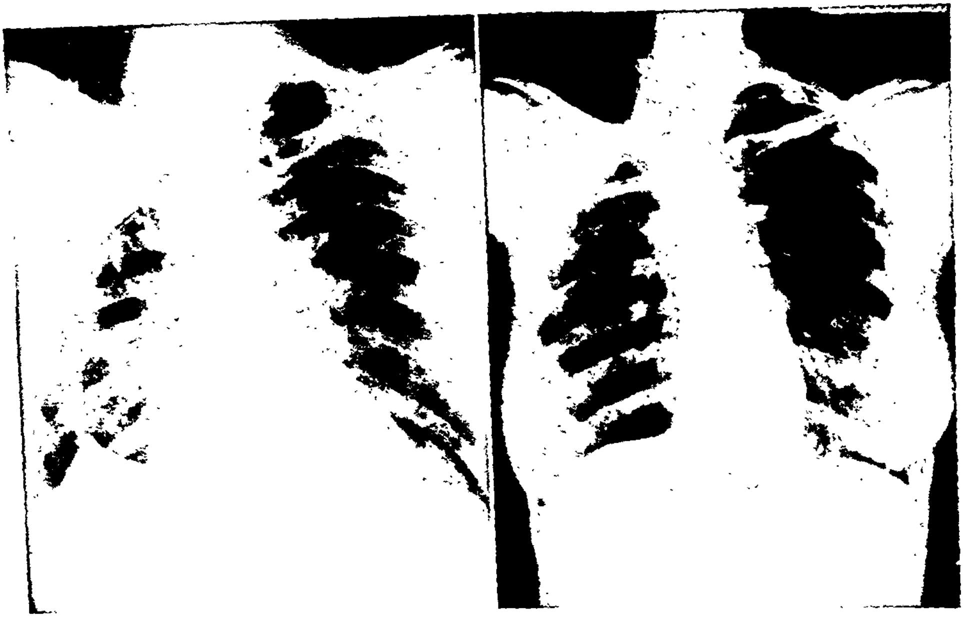
5. Earlier, more rapid obliteration of the pleural dead space is promoted, decreasing the hazards of bronchopleural fistula and empyema.

6. The concomitant operation eliminates one major procedure with its attendant risk and expense.

7. The presumed benefit of thoracoplasty is assured to the patient who might refuse a second operation.

8. Eliminating an additional operative procedure decreases the load on the hospital's surgical facilities and increases the number of patients who may be treated surgically in a given period of time.

Since we have susbstituted an intercostal incision for an incision through the periosteal bed of a long segment of resected rib, we believe that more function has been preserved by maintaining, to a greater extent, the integrity of chest wall structures over the nonresected portion of the lung. The differences are suggested, to a degree, by comparing the roentgenograph of a patient who had first a lobectomy (exposure through the bed of the fifth resected rib) with that of a patient who had a combined operation with use of an intercostal incision for exposure (Fig. 9). The comparison is not accurate nor are our impressions objectively substantiated; but it does seem that chest wall integrity and respiratory function are more dependent on intact, flexible ribs than on the loss due to dividing intercostal muscles.



A. *B.*

Fig. 9.—E. S., *A*, This patient, in March, 1948, had first a right upper lobe lobectomy, with a thoracotomy through the periosteal bed of the fifth rib. Nine days later a thoracoplasty, involving the first four ribs, was performed. *B*, V. K. Combined resection-thoracoplasty, September, 1950. Exposure by technique described in text, with thoracoplasty confined to graded segments of first four ribs.

Patients who have had prolonged pneumothorax therapy, continued or discontinued, and whose pulmonary tuberculosis is still uncontrolled, are frequently candidates for resection of residual caseating lesions and decortication of the remainder of the lung. In these patients one should not neglect the value of performing thoracoplasty at the same time in order to conform the size of the pleural cavity to the volume of the remaining decorticated portion of the lung (Figs. 3, 6, and 10).

Since thoracoplasties are sometimes advisable or mandatory following resections for nontuberculous suppurative or neoplastic disease it is obvious that some of these patients may also be candidates for the combined operation under consideration. We have performed thoracoplasty at the time of resection in a few such patients, seemingly to advantage; but the discussion here is concerned primarily with resections for pulmonary tuberculosis.

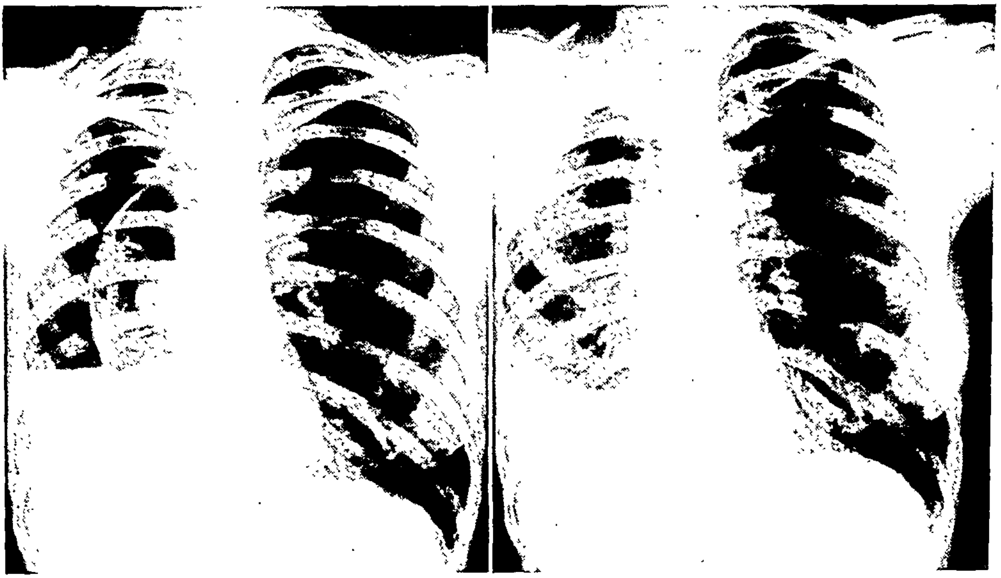


Fig. 10.—E. B., *A*, Right artificial pneumothorax since 1937. Pleural exudate positive for tubercle bacilli. Sputum was positive in July, 1949. *B*, Following decortication, resection of superior segment of right lower lobe (contained a cavity) and concomitant four-rib thoracoplasty (March, 1950). While very little lung was resected, the remainder of the lung, long collapsed, did not adequately fill the pleural cavity. Thoracoplasty was therefore performed at once.

In conclusion it may be stated that, with the exception of one patient who died, and one patient who required two subsequent thoracotomies for decortication and for closure of bronchopleural fistulae (but who now is convalescing satisfactorily), the combined operation of lobectomy or segmental resection with thoracoplasty has been well tolerated. Another patient had a bronchopleural fistula, but this responded promptly to intercostal tube drainage and her convalescence was otherwise uneventful (Fig. 5). The remainder of the patients appear to have made exceptionally smooth recoveries with less postoperative pain, less impairment of respiratory or muscle function and less morbidity than similar patients who have been treated by resection or thoracoplasty, alone or consecutively.

SUMMARY

1. The authors have presented their experiences with a group of thirty-eight tuberculous patients in whom pulmonary resection and thoracoplasty were performed at the same operation.

2. There was one postoperative death in the thirty-two patients who had upper lung resections, none in the six who had pneumonectomy and thoracoplasty.

3. The combined operation seems to be well tolerated and the incidence of postoperative complications is low.

4. A number of advantages of the combined procedure over separate operations has been adduced from our experience and from the reports of others: a. Use of a modified thoracotomy incision and less extensive rib resection make for adequate exposure and for a better functional result than in the usual postresection thoracoplasty. b. The thoracoplasty collapse can be accurately conformed to the remaining portion of the lung before it becomes distended and adherent. Subtotal resection, in preference to pneumonectomy, can be undertaken with more confidence. c. The pleural dead space is promptly obliterated by thoracoplasty, decreasing the hazard of bronchopleural fistula and empyema. d. Elimination of one major procedure and its attendant risk is important to the patient and to the hospital, and patients with resection who might refuse a second operation are assured the protection of thoracoplasty.

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DISCUSSION

DR. PAUL C. SAMSON, Oakland, Calif.—Since apparently I am one of the early group of surgeons to concern myself consistently with concomitant operations, I feel that I should stand up and be counted so that if there are any who are sharpening axes I can offer another broad pair of shoulders to work on.

I enjoyed Dr. Conklin's presentation very much and his experiences parallel ours. As for our technique, we have left the first rib in and have peeled the pleura down from beneath the first rib, believing that there would be less deformity than if the first rib was removed. I became interested in combined procedures in 1949, following Skinner's report, and it seemed to me that lobectomy-thoracoplasty was more important, perhaps, than pneumonectomy-thoracoplasty. In the former, the tailoring of the chest wall to fit the remaining lobe seems to be rational and there is certainly less bronchial distortion than if phrenic nerve paralysis and/or pneumonoperitoneum is used to fill the space. I would only emphasize that the combining of thoracoplasty and resection is but one facet of the entire treatment problem in these patients. In addition to proper preoperative evaluation, including bronchoscopy with right-angle telescopic examination of the upper lobe bronchus, the proper timing of operation and the intelligent use of antimicrobial therapy, I stress the great importance of strict bed rest, preferably for six months following resection. In the main, the majority of the delayed complications encountered in our entire resection program have been in patients who insisted upon relatively early ambulation and return to work.

Since January, 1949, Dr. Dugan and I (or our associates) have performed fifty-five concomitant resections with thoracoplasty. There was one immediate postoperative death, a 1.8 per cent mortality, and one delayed death, sixteen months after operation. There were forty lobectomy-thoracoplasties and fifteen pneumonectomy-thoracoplasties. There were no immediate postoperative complications of major concern in the entire series: No wound infections, no empyemas, and no spreads. There was one questionable reactivation which cleared up entirely in a few weeks. Of the fifty-three living patients, fifty are sputum-negative as of recent report.

A year ago I presented preliminary data on 26 of these patients before the California Trudeau Society and this report recently has been published as Dr. Conklin indicated.* The present status of these twenty-six patients whose operations were performed from fifteen to twenty-seven months ago is of interest. There were seventeen lobectomy-thoracoplasties with the one postoperative death previously mentioned. Of the sixteen living patients, fifteen are sputum-negative and working—a 94 per cent conversion rate; the sixteenth had active contralateral disease at the time of operation, which cleared, but later on activity developed a cavity and he is still positive. Of the nine pneumonectomy-thoracoplasties, eight are living and negative. Of these, six are at home and working; two developed a late basal empyema and are still in the hospital. The ninth patient was discharged immediately following surgery with a clear contralateral lung. At home, activity could not be restricted. Following a cold five months postoperatively, she developed active disease and died of progressive pulmonary tuberculosis sixteen months following operation.

DR. JAMES E. DAILEY, Houston, Texas.—My shoulders are not quite as broad as Dr. Samson's, but I could be included with the group subscribing to his opinions, if necessary.

For some time my associate Dr. Barkley and myself have felt, as Dr. Conklin has so excellently shown you today regarding thoracoplasties and resections, that the best time to perform this thoracoplasty would be simultaneously with the resection. We started a series of these cases, the first one in April, 1949. It is not as large a series as those that have been presented to you; it consists of only twenty-three cases, the youngest 6 years old and the oldest 56. Three of these were total pneumonectomies and twenty were lobectomies. It is not quite clear to me why Dr. Conklin advises against the combined procedure for lower lobe resections, inasmuch as we have felt that the elevation of the diaphragm was not quite sufficient to obliterate the space. Of the twenty lobectomies in our series sixteen were for lower lobe and four were for upper lobe disease. We had no deaths in the series and all but two immediately converted their sputum and have remained negative since then. The two that still have positive sputa had contralateral cavities and further procedures are contemplated for them.

If I had slides with me I believe I would be able to demonstrate the surprising lack of deformity in these lower lobe thoracoplasties. The effectiveness of the thoracoplasty in obliterating space cannot be appreciated very much by the postero-anterior and lateral roentgenographs, but roentgenographs in the oblique view show the depression in the thorax very well. It is possible that use of a plastic sponge might prove better for obliteration of this space after resection. We have had no experience with this. However, if thoracoplasty is chosen we would certainly subscribe to doing this procedure at the same time as the resection.

DR. CHARLES W. LESTER, New York.—I also am in favor of this procedure of thoracoplasty at the time of pneumonectomy or lobectomy. We first started using it some years ago in children because we thought that over-distension of the remaining lung produced respiratory difficulties in some instances, and we subsequently modified it to use in adults.

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*Samson, P. C., Dugan, D. J., and Harper, H. P.: Upper Lobe Lobectomy and Concomitant Thoracoplasty in Pulmonary Tuberculosis, California Med. 73: 547-549, 1950.

(Slide.) This is the first case in which we used it; an 8-year-old girl who had a pneumonectomy for bronchiectasis. She has long posterior stumps and the first and second ribs are left in. It is quite adequate for an 8-year-old child but we do not think it would work with adults.

(Slide.) We therefore modified the procedure as shown here. We enter the chest through the resected fifth rib after the resection is completed—and this thoracoplasty procedure takes only about five minutes and does not add to the shock of operation—we take half the fourth rib, one-third of the third, and cut the second, leaving long posterior rib ends extending at least a centimeter beyond the transverse processes. If it is an upper lobe lobectomy that is all that is done: if it is a pneumonectomy we take half the sixth, and also cut the seventh in the same way the second is cut.

(Slide.) This is a two-year result in a 14-year-old girl who had a left pneumonectomy for tuberculosis. Two years after operation she was studied by Dr. Cournand, and she has only 16 per cent residual air, which is remarkably low. She has, however, what appears to be a little anatomic distention, although physiologically she has none.

In doing these pneumonectomies we feel that excess fluid should be aspirated early and often so as to permit the collapse to take place. If this is not done the chest wall will not collapse sufficiently.

(Slide.) This is a 23-year-old girl who had a destroyed right lung which also, as we found out afterward, had carcinoma in it. It was a long gruelling operation but the thoracoplasty was done at the end of it. The thoracoplasty procedure produced a fair amount of collapse, but there was so much blood and fibrin that we could not aspirate it properly, although at two months it looked like a very good result.

(Slide.) At eight months when the fluid absorbed, her heart had moved to the right side and the left lung had gone back to the position it occupied before operation.

(Slide.) This man had a pleuro-pneumonectomy, and shows another advantage to this thoracoplasty procedure. If you have to do a second procedure, that is, if you have to do more thoracoplasty, you can go in through an axillary incision. There is little blood loss and no shock to that procedure and if you have to close a broncho-pleural fistula there is plenty of muscle in the axillary fold to use as graft. This man opened his fistula eight months after operation. The thoracoplasty was completed through an axillary approach and the broncho-pleural fistula was closed in the same operation.

(Slide.) This man had decortication of the lower lobe and lobectomy of the upper lobe. The modified thoracoplasty was done at the same time and the chest was drained. The procedure allowed a better collapse and he has good pulmonary function.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—Dr. Conklin has proposed a most worth-while subject for consideration. I should like to bring out two points: first, I do not believe that the combined procedure should be used routinely in all cases. There may be added risk. We have used it in approximately half the patients being treated for tuberculosis with resection in which we felt that ultimately they would need some reduction in thoracic volume. We have not considered it wise to do a simultaneous thoracoplasty if the operation has been a prolonged one or if the general condition of the patient at the conclusion of the resection would make it seem that this added procedure might increase the risk.

The second point is that if one is attracted by the single stage procedure, I think it is well to maintain the integrity of the chest. It is possible to retailor the chest, maintain rigidity and prevent danger of paradoxic chest-wall movement.

(Slide.) The first step in the method we employ is to divide ribs two, three, *four*, and five anteriorly from within the chest.

(Slide.) The second step divides these ribs posteriorly and separates the periosteum from the under surface of the first rib.

(Slide.) The third step removes appropriate posterior segments of the *second, third,* fourth and fifth ribs.

(Slide.) In the final step, the liberated and shortened plate of remaining segments of ribs are swung downward and inward. They are securely fastened to posterior rib stumps. The second rib is usually fastened to the fourth stump, the third to the fifth, etc.

A roentgenograph shows the degree of chest-wall shift obtained by this type of modified thoracoplasty.

The fact that the resection and retailoring can be done in one stage effects great saving in comfort and hospital expense. We have lost only one patient, a diabetic, in whom the combined operation was performed. It was difficult to tell whether or not the added procedure contributed to the outcome. I would, however, like to issue a warning to the effect that if one contemplates using the combined procedure, the cases should be carefully selected.

DR. W. D. KELLEY, Norwich, Conn.—It is encouraging to know that there is still an interest in the surgical treatment of tuberculosis. In spite of the advent and rebirth of such a panacea as BCG we still have a problem. As you know, the tuberculosis death rate throughout the country is something like forty per one hundred thousand population, and even in the small state of Connecticut and other places where we think that tuberculosis has a fairly well-organized program, the death rate is still about thirty per one hundred thousand. So we still have a problem in treatment and I think we should emphasize surgical treatment of this disease. It is interesting to note also that there is such a wide variety in plans of treatment; in fact, it is almost astounding that there should be so many different types of surgical treatment for practically identical types of disease. I wonder if this is not the right time to question whether or not we should not try to come to some standardization of surgical treatment.

As a matter of completeness, I would like to mention our statistics in one surgical unit in Connecticut in relation to resections for tuberculosis. We have a series now of eighty-two cases of resections, including pneumonectomies, lobectomies, and segmentals. There are thirty-one cases of lobectomy, twenty-nine cases of pneumonectomy in this group. In connection with this I would like to say that we have tried the combined operation in three cases; one a lobectomy and two a pneumonectomy, and we found in two of the three cases that there was a considerable increase in morbidity and for that reason we have not continued with the combined operation of thoracoplasty and pneumonectomy.

DR. LAURENCE MISCALL, New York.—In our experience at Triboro Hospital we have not combined resection therapy with thoracoplasty. If we intend to use thoracoplasty it is completed usually as a primary procedure, after which resection has been carried out at a later date. Yet, in certain cases, we have found combined procedure advisable. In these we tailored the thorax by a method which the following slides illustrate. Resection has been combined with mobilization of the apical pleura, which has been turned down over the lung and sutured to the lower margin of the line of incision. This has created an extrapleural space.

(Slide.) On this first plate taken immediately after operation the dots outline the air-filled space above the right middle and lower lobes after removal of the right upper lobe.

(Slide.) This second slide shows that effusion into this space has occurred rapidly. It is clearly demonstrated by the small fluid level at the top of the space.

(Slide.) This next plate taken eight days after operation shows that the complete filling of the space by fluid has compressed the lung and helped to rapidly obliterate the pleural space by bringing it into contact with the lung.

(Slide.) This plate taken five days later shows the ease with which pulmonary expansion can be regulated by aspiration of fluid from the extrapleural collection.

(Slide.) In this lateral view one can see that the right lower and middle lobes have selectively expanded; that the pleural tent parallels the margin of the re-expanded lobe and that the fluid above has decreased.

(Slide.) This plate indicates the ability of collections to persist and organize. At this time, seven months after operation, the pleural tent is equivalent to the four- or five-rib thoracoplasty. The chest wall has not been disturbed except for the excision of one rib. He was always able to raise secretions well after operation. He has maintained a straight spine and has been quite comfortable. It is possible that these factors have better preserved respiratory function than that observed with similar collapse from thoracoplasty.

(Slide.) This is a plate taken on Mar. 19, 1948, ten days after resection of a left upper lobe for tuberculosis. The large extrapleural hemopneumothorax which can be clearly seen was aspirated on three occasions.

(Slide.) On this plate of Jan. 5, 1949, ten months after operation, one can see the left lower lobe has re-expanded to the level of the fifth rib. Note that the spine has remained absolutely straight and this corresponds to a thoracoplasty of similar degree.

(Slide.) Here is a plate on another patient who had a left upper lobectomy for a tuberculous cavity seen well on the plate on the right.

(Slide.) I am sure, following the report of Dr. Chamberlain, that segmental resection would be preferred now. This (slide) lobectomy, however, was combined with a pleural tent. The effusion into the extrapleural space was aspirated carefully to control expansion. The plate on the right three years later illustrates the definite restriction of overexpansion of the remaining lower lobe.

Our experience with the pleural tent leads us to believe that it is definitely superior to thoracoplasty when one wishes to tailor the thorax at the same time when resection is completed.

DR. LEO ELOESSER, San Francisco.—Pardon me if I take a few words to call attention to something that perhaps you all know. I did not gather from the speakers exactly how they did the thoracoplasty. But through all these very large chest incisions it is quite easy to remove the desired amount of ribs, working from the inside of the pleural cavity, either transpleurally or, if the occasion dictates, subpleurally by pushing the pleura aside.

DR. JOHN M. CHAMBERLAIN, New York.—I want to take a minute to add a word of caution about the use of this combined procedure. On Dr. Berry's service at Bellevue and the other services with which I am associated, we have used the combined procedure in sixty patients. I am sure Dr. Conklin will quickly and readily admit that the postoperative course of these patients is a little more hazardous, and that perhaps the morbidity is increased, because of the paradoxical chest, as Dr. Overholt has pointed out. One other point: In doing these operations it is frequently necessary to go into the extrapleural plane in order to mobilize the lobe. In such cases, when one follows the resection with a thoracoplasty, there are only ribbons of periosteum and intercostal muscle to fall in and obliterate this space. This defect allows the pleural cavity to be in communication with the subscapular collection of fluid and one must therefore deal with a larger hemothorax postoperatively. As a result, I believe the management of these cases requires a great deal more attention. For these reasons, we now enter the pleural cavity through the fourth rib instead of the fifth and then take short segments of ribs three and two. In this way we are able to reduce to a minimum the paradoxical motion. We then mobilize the apex of the lung by incising the periosteum beneath the first rib, just as Dr. Samson does.

DR. CONKLIN (closing).—I thank the numerous discussors and hope that at any future appearance before this Association I may have Dr. Samson's broad shoulders to lean upon. Dr. Dailey mentioned the use of thoracoplasty in conjunction with lower lobe resection. We have thought that the diaphragm does elevate sufficiently to fill the space, and have thought that performing thoracoplasty in these cases displaces the upper and middle lobes in such a manner that it may kink the bronchus or otherwise affect efficiency. The use of a plastic sponge in lobectomy does not seem to me a practicable solution, and certainly any foreign body does not help with the hazards of empyema and bronchopleural fistula.

We have used this procedure, of course, in carefully selected nontuberculous cases, suppurative disease and neoplastic disease. So far as aspiration of fluid is concerned following resection and thoracoplasty, as mentioned by Dr. Lester, this is done in our cases according to indications, such as displacement of the mediastinum, and so on. From the paper of Drs. Iverson and Skinner I understand that they always drain the pleural cavity following pneumonectomy and thoracoplasty, and I think in these cases the use of drainage following pneumonectomy is better justified than when no thoracoplasty is performed. Drainage will promote obliteration of the pleural space and there will not be excessive mediastinal displacement as a result of drainage.

We do not use the combined procedure routinely. In other words, as Dr. Overholt mentioned, if the patient's condition does not seem entirely satisfactory when the resection is completed, a contemplated thoracoplasty should be deferred. However, I would like to point out that, particularly with the type of technique we are using, it prolongs the operation very little. Ordinarily we resect one, and very frequently two, of the ribs to be resected before entering the pleural cavity, and that leaves only the uppermost two ribs to be resected at the end of the procedure, and this can be accomplished very rapidly. Dr. Samson carefully timed his cases and found that thoracoplasty added an average of only sixteen minutes to the entire combined procedure and I feel that with a more limited type of thoracoplasty, such as we perform, it averages even less.

Dr. Eloesser asked about the approach to the ribs, whether from the inside or outside. We have on occasion used an intrapleural approach to the ribs and have found it advantageous at times to divide the costal cartilage of the first or second rib from within the pleural cavity with a scalpel. However, we prefer to use the extrapleural approach, dividing subscapular muscles, feeling that in this way the periosteum will drop down against the mediastinum and that the benefit of the thoracoplasty will be promptly achieved.

REVISION THORACOPLASTY

A REVIEW OF TWENTY-SEVEN CASES

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THE literature on revision thoracoplasty is somewhat confusing. In 1936, Kinsella[1] advised extraperiosteal gauze packing with revision except in those patients with small cavities who had inadequate first operations. By use of packing, he was able to close eleven of thirteen cavities. Without packing, he was successful in twelve of twenty-three cases. Alexander[2] recommended revision for unsuccessful, inadequately performed thoracoplasties. He felt that the operation was not indicated after a thoracoplasty in which the transverse processes and adequate lengths and number of ribs had been resected. Revision in his hands was successful in twelve of a total of fifteen thoracoplasty failures. Welles,[3] being of the opinion that the real cause of thoracoplasty failure lay in the lesion within the lung, did not recommend revision. He attempted to close the cavities in six thoracoplasty failures by resecting regenerated ribs and transverse processes, and was unsuccessful in all. Weinstein[4] recommended that revision include resection of ribs over the cavity, shortening of adjacent transverse processes, and mobilization of the lung from the vertebral bodies. By this method he was able to convert the sputum of 54.6 per cent of twenty-two patients whose first thoracoplasties had been adequate. Judd[5] recommended plication of the lung on itself to close cavities under thoracoplasties which had been adequate. This method was successful in converting the sputum of 4.35 per cent of twenty-three cases. Hochberg, Fink, and Denize,[6] after reviewing a series of eighty-nine cases of revision thoracoplasty, concluded that revision was most successful in patients whose first thoracoplasty had been inadequate. They were able to convert the sputum of about one-half of the latter group as compared to about one-fourth of their total group.

The twenty-seven cases of revision thoracoplasty presented in this article were consecutively operated upon at Uncas-on-Thames between July, 1946, and January, 1951, during which time 278 cases of thoracoplasty of all types were performed. There were one operative mortality (3.7 per cent) and two late mortalities (7.4 per cent). Two patients received inadequate revisions without resection of adequate lengths of transverse processes, and both failed to develop a negative sputum. Twenty-four patients had what were considered adequate revisions.

In Table I are summarized the results of revision according to accepted criteria for judging adequacy of the first thoracoplasty. Thoracoplasties were considered adequate if the first two ribs had been completely or almost com-

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TABLE I. FOLLOW-UP ON TWENTY-FOUR CASES OF SATISFACTORY REVISIONS

FIRST THORACOPLASTY	NUMBER	SPUTUM CONVERTED TO NEGATIVE	CAVITY CLOSED		CONTRA-LATERAL CAVITY PRESENT
			BY LAMINAGRAM	BY PLAIN FILM	
I. Unsatisfactory Operation	18	10	6	6	3
A. Inadequate Transverse Processes	9*	6*	4*	5	3
B. Inadequate Transverse Processes and Inadequate Number of Ribs	5	4	2	1	0
C. Inadequate Transverse Processes and Delay Between Stages	1	0	0	0	0
D. Inadequate Transverse Processes, Delay Between Stages and Inadequate Number of Ribs	1†	1	1	0	0
E. Inadequate Number of Ribs	2	0	0	1	0
F. Inadequate Lengths of Ribs	0	0	0	0	0
II. Satisfactory Operations	6	3	2	1	1

*According to death certificate, one of these patients died of "tuberculosis and pneumonia," about two years after revision. Last recorded sputum, one year after revision, was negative. Last available roentgenogram showed no cavity one and one-half years after revision.

†One of these patients died about two and one-half years after revision from carcinoma of the lip. Roentgenogram before revision did not reveal definite cavity.

pletely resected, if the transverse processes had been removed flush with the laminas, if sufficient lengths and numbers of ribs had been resected to collapse the lung well below the cavitary disease, and if a delay of more than three to four weeks had not occurred between stages. A revision operation was considered adequate if it corrected the defects in the first operation. Anterior thoracoplasties were performed according to the method of Haight[7] in all but three of the twenty-four patients summarized in the table.

Reference to Table I will reveal that roughly one-half of the patients who had satisfactory primary operations benefited by revision. In contrast, about two-thirds of the group who had unsatisfactory primary operations benefited

TABLE II. RESULTS OF REVISION ACCORDING TO ROENTGENOGRAM APPEARANCE OF DISEASE BEFORE FIRST THORACOPLASTY

X-RAY APPEARANCE	NUMBER CASES	NUMBER CAVITY CLOSED	NUMBER SPUTUM CONVERTED NEGATIVE	NUMBER WITH CONTRALATERAL CAVITY PRESENT
Giant Cavity	5†	4	4	1
Dense Disease	3*	3*	2*	1
Giant Cavity and Dense Disease	1	0	0	0
Small Thin-Walled Cavity with Infiltration of Slight Density	10	6	5	0

*According to death certificate, one of these patients died of "tuberculosis and pneumonia," about two years after revision. Last recorded sputum, one year after revision, was negative. Last available roentgenogram showed no cavity one and one-half years after revision.

†One of these patients died about two and one-half years after revision from carcinoma of the lip. Roentgenogram before revision did not reveal definite cavity.

by revision. If the group with inadequate removal of transverse processes at primary operation is studied, it will be seen that from three-fourths to four-fifths of these benefited by revision.

Table II summarizes the results of adequate revision according to the roentgenogram appearance of disease prior to the first thoracoplasty. The preoperative roentgenograms were available for review in nineteen cases. In each instance, cavitary disease was apparently localized to the upper lobe. It will be

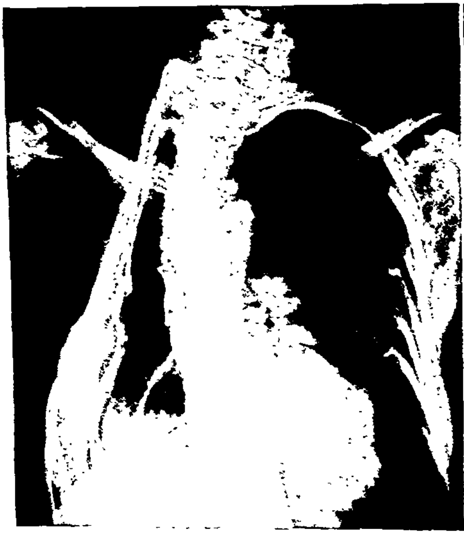

Fig. 1.

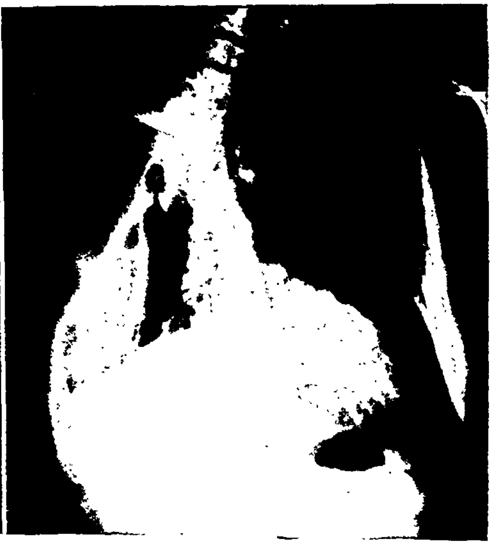

Fig. 2.

Fig. 1.—Chest roentgenogram of typical case in which transverse processes had not been resected at time of original thoracoplasty.

Fig. 2.—Roentgenogram of same patient following revision thoracoplasty. Appropriate transverse processes and regenerated ribs have been removed.

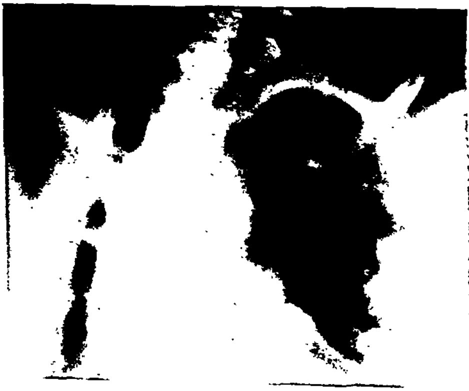

Fig. 3.

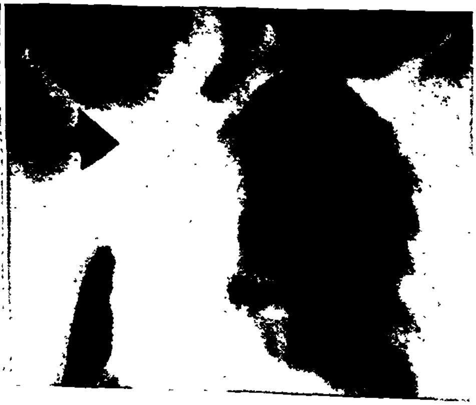

Fig. 4.

Fig. 3.—Laminagram of same patient just before revision thoracoplasty. Cavity clearly visible just anterior to transverse processes.

Fig. 4.—Laminagram following revision. Arrow points to region where cavity had been clearly visible but is indefinite. Case is still classified as unsatisfactory result even though sputum is now negative.

noted that there was no significant difference in the response of the categories listed to secondary collapse. One might, therefore, conclude from a study of the two tables that mechanical factors are of more importance than pulmonary pathology (as seen by roentgenogram) in determining the results of thoracoplasty.

Streptomycin therapy was employed in conjunction with revision in four patients, only two of whose operations were adequate. The latter two patients were the only ones in this group to develop negative sputum, which would seem to substantiate the opinion that streptomycin is not a substitute for satisfactory collapse.

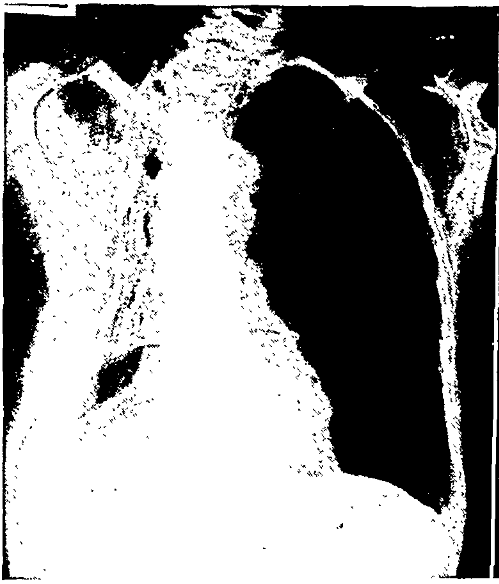

Fig. 5.

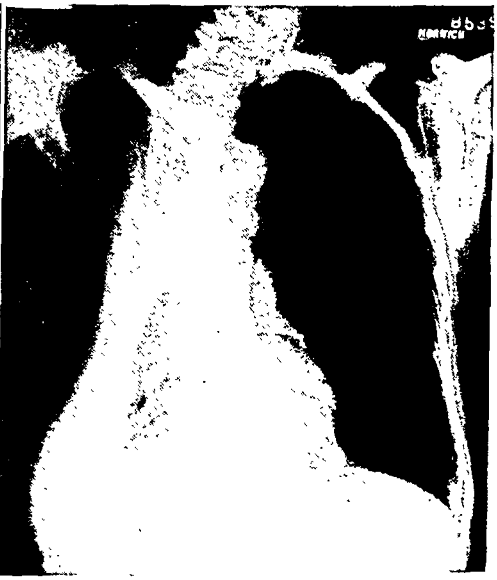

Fig. 6.

Fig. 5.—Chest roentgenogram showing inadequate removal of transverse processes and anterior rib ends. Diseased lung not satisfactorily collapsed.

Fig. 6.—Roentgenogram of same patient following anterior and posterior revision thoracoplasty showing satisfactory collapse.

Ventilatory function studies were performed on only two patients prior to and following secondary operations. In one instance, the removal of five regenerated ribs, a portion of one additional rib, and an anterior stage diminished the maximum breathing capacity from 92 to 75 liters per minute. In the other case, with removal of eight transverse processes and regenerated ribs and an anterior stage, the maximum breathing capacity changed from 64 to 58 liters per minute. In general, it can be said that respiratory function was not impaired to any great extent by revision thoracoplasty.

It was formerly our custom to resect all or most of the regenerated ribs shortly before or at the time of thoracotomy for pulmonary resection. Four of the patients included in this report had revision and thoracotomy without resection because of technical difficulties. In each case, the lung was freed to a variable degree. Two patients, who had technically adequate revision operations, have negative sputum and no evidence of cavity. The other two had technically inadequate revisions, and showed persistent cavity and positive sputum. At least in this small group, partial pneumonolysis did not appear to be beneficial.

SUMMARY AND CONCLUSIONS

1. Twenty-seven cases of revision thoracoplasty are reviewed. There were one operative and two late deaths.

2. Revision operation is most successful following inadequate thoracoplasty in which the presence of transverse processes seems to be a significant factor interfering with adequate collapse.

3. The type of disease as noted by roentgenogram in this series did not appear to be a determining factor in the success of revision thoracoplasty.

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LIPOSARCOMA OF THE MEDIASTINUM

Report of a Case With Associated Lipomas of the Mediastinum and Subcutaneous Tissues

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THE literature contains no record of a patient who exhibited the simultaneous presence of a lipoma and a liposarcoma of the mediastinum. This apparently unique association justifies the reporting of a single case.

Liposarcoma is a rare tumor.[1-5] The most common sites of origin are the fascial planes of the thighs and the retroperitoneal tissues. Lipomas, on the other hand, are among the most frequently encountered of all neoplasms. Although seen in many locations, they usually occur in the subcutaneous tissues. The mediastinum is a rare site for either of these tumors.[1, 2, 6-11] The number of mediastinal lipomas reported in the literature comprises a very small group. Less than fifty have been recorded in all intrathoracic locations.[11] Many of these were lesions of the dumb-bell type with a portion of the tumor situated in the neck or chest wall and connected with the intrathoracic component by a stalk or pedicle. Lipomas confined solely to the mediastinum are unusual. The rarity of mediastinal liposarcomas is attested by the fact that a search of the literature reveals only five solitary tumors of that type located in the mediastinum.[2, 6, 12-14] Five cases of multiple liposarcomas have been reported,[15-19] and in at least two of these the mediastinal organs or structures as well as other more common sites were involved.[15, 19]

Benign lipoma and its malignant counterpart, liposarcoma, are very rarely found in combination. Indeed, we have been able to find in the literature only three previously recorded instances of such an association, namely, the case reported by Farbman[20] and the two cases recorded by Starkloff, Saxton, and Johnson.[21]

CASE REPORT

The patient, a 55-year-old Naval officer on active duty, was admitted to the U. S. Naval Hospital, St. Albans, L. I., N. Y., on Nov. 27, 1950, because of the discovery of a large, right superior mediastinal mass by routine chest roentgenogram taken at the time of his annual physical examination. He voiced no complaints except for a mild "cigarette cough" which had been present for many years. During recent months the cough had been harsh, dry, and somewhat more annoying than previously. There was slight huskiness of the voice. The patient attributed both the increased cough and mild hoarseness

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From the U. S. Naval Hospital, St. Albans, N. Y.

The opinions or assertions contained herein are those of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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to a strenuous program of public speaking which he had recently been called upon to undertake in connection with his official duties. In the past few weeks he had noticed a tendency to tire somewhat more easily than formerly. Although still able to participate in sports, such as golf and tennis, he had observed increasing shortness of breath after vigorous exercise. He had no chest pain, expectoration, or weight loss. His past medical history revealed no relevant facts. In 1941 he was hospitalized for a few days for acute cystitis. Five years prior to admission he had been discovered on routine electrocardiogram to have a right bundle branch block. This condition persisted but had never given rise to symptoms, and apparently was of no serious clinical significance. There were no facts in his family history pertinent to his present illness.

Complete physical examination showed few noteworthy findings. He was observed to have an intermittent, harsh dry cough. The voice was slightly husky. Rather prominent spider hemangiomata were present over the upper sternal area and on the right upper chest anteriorly. The patient's temperature, pulse, and respiratory rates were normal. His blood pressure was 140/90, and equal in the two arms. There were no abnormal physical signs noted on examination of the heart and lungs. Abdominal examination was essentially negative except for a somewhat irregularly shaped mass approximately 8 by 4 by 3 cm. in size, which could be seen and felt beneath the skin in the left lower abdominal quadrant. On palpation this mass appeared to be lobulated. It was not tender and was freely movable, being firmly attached to neither the overlying skin nor the underlying tissues. The physical findings were entirely consistent with those commonly exhibited by a lipoma. The extremities were normal. There was no peripheral lymphadenopathy. Neurological examination was negative. Indirect laryngoscopy showed normal appearing vocal cords whose motion was not impaired.

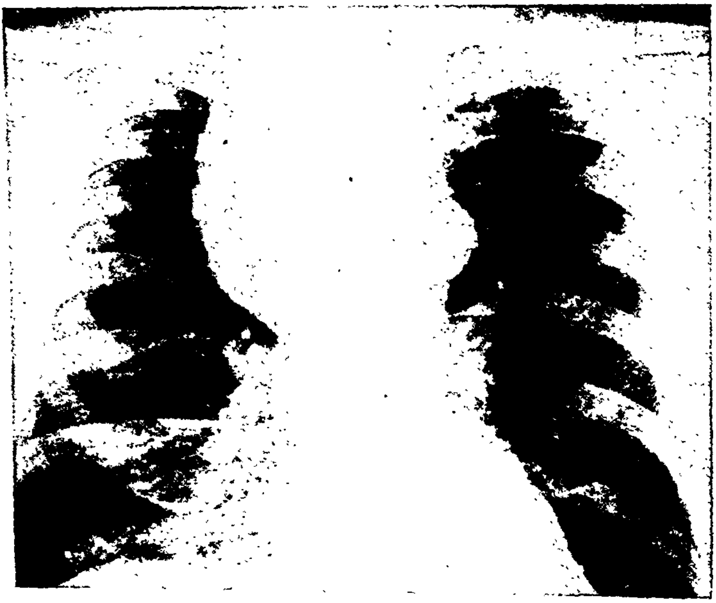

Fig. 1.—Posteroanterior chest roentgenogram taken just prior to admission showing the large superior mediastinal tumor projecting into the right hemithorax.

Routine laboratory studies, including liver and renal function tests, were within the limits of normal. The Kahn test was negative. Posteroanterior (Fig. 1) stereoscopic and right lateral (Fig. 2) chest roentgenograms showed an ovoid mass of homogeneous density, about 14 by 8.5 by 7 cm. in size, which projected to the right from the superior mediastinum. The mass extended from the thoracic inlet to the level of the right hilum.

Stereoscopically and in the lateral projection it was seen to be situated in the upper posterior and mid-mediastinal areas. The tumor was perfectly regular in outline and very sharply demarcated. Its density was perhaps slightly less than one might anticipate from a mass its size. The mediastinum was not displaced, and the lung fields were clear in all areas other than that obscured by the projecting mediastinal mass. A photoroentgenogram made one year previously was available for comparison (Fig. 3). It showed, in retrospect, a clearly visible right paratracheal superior mediastinal tumor which was overlooked when the miniature film was originally read. In the interim of approximately one year the mass had increased at least tenfold in size. No pulsations of the tumor were detected on fluoroscopy, and it was observed that the mass did not move with the lung on respiration.

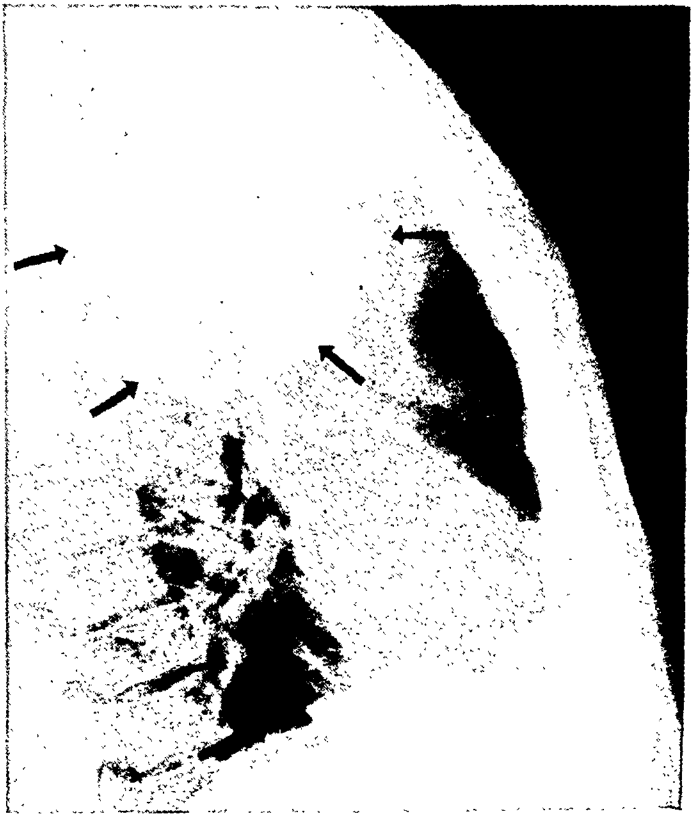

Fig. 2.—Right lateral chest roentgenogram taken a few days prior to operation which shows the central and posterior location of the tumor.

Angiocardiography (Fig. 4) showed that the lesion was definitely extrapulmonary in location. The vascular pattern was normal in the right lung. This procedure not only proved that the mass was not an aneurysm or other vascular lesion, but also showed that there was no serious compression of the great vessels in the mediastinum. Bronchoscopy showed no mucosal lesion in the tracheo-bronchial tree. However, there was marked evidence of compression of the lower trachea and right main stem bronchus, apparently caused by an extrinsic mass on the right. The right lateral wall of the lower half of the trachea was displaced medially. The main carina was blunted and foreshortened in the anteroposterior diameter. The right main stem bronchus was reduced approximately 50 per cent in caliber, and both this bronchus and the lower half of the trachea showed considerable fixation. Although there was definite diminution of the normal mobility of these structures, fixation was not of the rigid type. The mucosa of the distal trachea and right main stem bronchus was thrown into longitudinal folds or furrows, as if the structures were being squeezed from without. A good view could not be had of the right upper lobe orifice due to the reduction in mobility of the trachea and right main stem bronchus. However, as seen tangentially, it was noted that the upper lobe bronchial lumen was also markedly reduced. The right lower bronchial tree and the entire left side were normal in all respects. The right upper lobe bronchus was lavaged and the aspirate collected for Papanicolaou smears, which were reported as suspicious for malignant cells, Class III. Several subsequent sputum specimens examined by the same technique were negative for malignant cells.

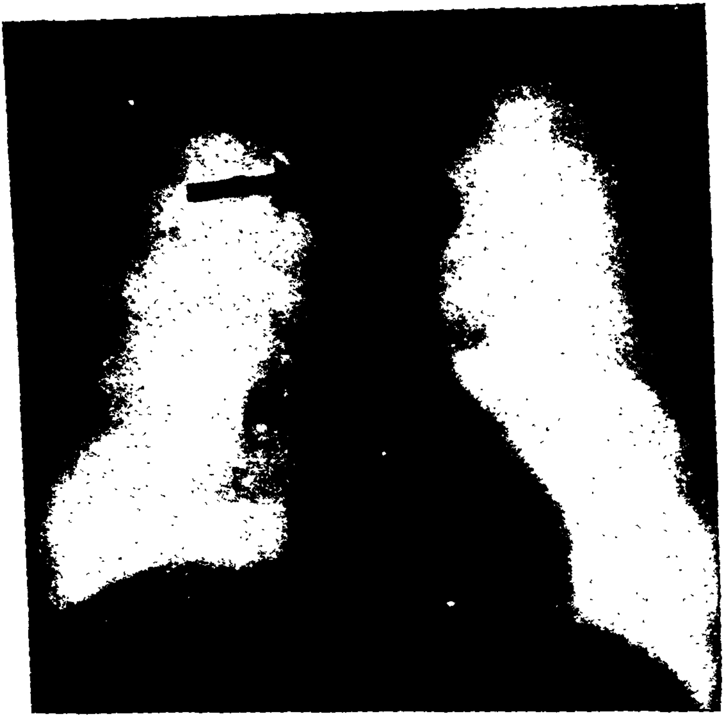

Fig. 3.—Enlargement of a 35 mm. photoroentgenogram made one year prior to admission. Although overlooked when the miniature film was read, the mass was clearly visible at that time.

The subcutaneous mass was removed from the left lower abdominal quadrant on Nov. 27, 1950. Grossly it appeared to be a typical lipoma. The pathologic diagnosis was "lipoma."

Right posterolateral thoracotomy was performed on Dec. 2, 1950. The right fifth rib was resected and the pleural cavity entered by an incision in the bed of the resected rib. A free pleural space was encountered except for a small area over the tumor mass laterally to which the mediastinal surface of the right upper lobe was lightly adherent by fine, easily separated cobweb adhesions. The right lung was normal on inspection and palpation. Examination of the right superior mediastinum established the presence of *two* tumor masses. The larger of these, the size of a large grapefruit, was confined to the mediastinum and completely covered with pleura on its presenting surface. It extended principally into the right chest but, as was demonstrated during its removal, also projected slightly into the left pleural space. In addition, it was found to extend through the right superior thoracic aperture into the lower cervical region. The inferior margin of the larger mass rested on the smaller tumor to which it was somewhat adherent. The smaller mass in turn abutted inferiorly on the lung root and lay in the crotch between the superior vena cava and the azygos vein. The azygos vein crossed the lower pole of the smaller lesion and was considerably compressed by it. The posteromedial aspect of the smaller neoplasm lay along the lateral tracheal wall and inferiorly nestled in the angle between it and the superior surface of the right main stem bronchus. Viewed from the side, the masses were seen to occupy a central and posterior position in the superior mediastinum, but they did not extend into the costovertebral gutter. Both the right phrenic and vagus nerves coursed in their usual relationship to each other over the surfaces of the masses and were drawn as tight as bowstrings as a result of lateral displacement by the larger tumor. The pleura over the tumors was split in the vertical direction, and the masses were found to be well encapsulated. An excellent line of cleavage was easily established outside the tumor capsules, and they were readily freed by blunt finger dissection. Each was enucleated intact without difficulty. During the course of the dissection the larger mass, somewhat lobulated in contour, was found to extend across the midline anterior to the trachea and to project slightly into the left chest. There was also a prolongation which traversed the superior pulmonary sulcus and extended for a short distance into the root of the neck on the right. The smaller mass had no identifiable vascular pedicle. The larger tumor was completely freed except for one small area which was seen to contain a slender vascular pedicle no larger than one-half the diameter of a lead pencil. After the pedicle had been dissected out for a suitable distance it was ligated and severed 2 or 3 cm. distal to the mass. When the two tumors had been removed careful examination of the entire right mediastinum showed no residual tumor masses. The chest was closed in the usual manner.

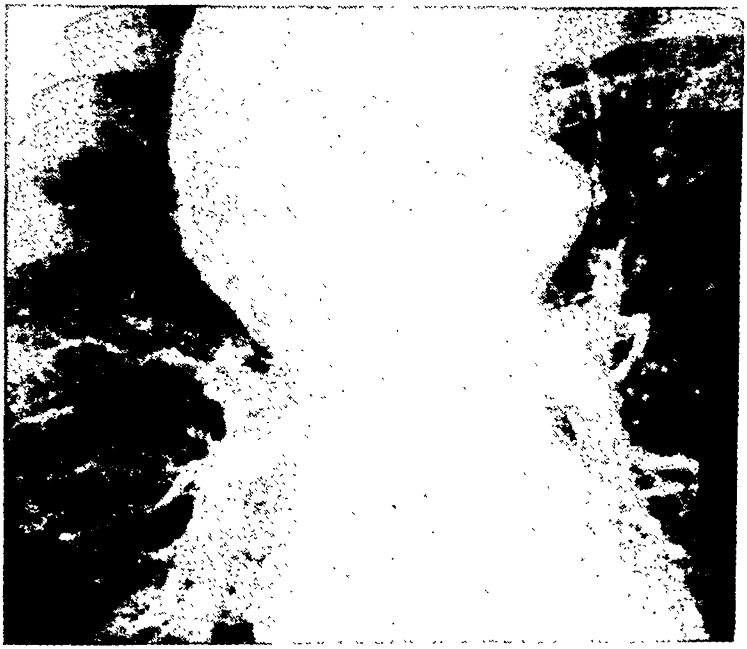

Fig. 4.—Angiocardiogram made shortly before operation. This demonstrated that the lesion was not an aneurysm and showed a normal vascular pattern in the right lung.

The pathologic report was as follows:

Macroscopic: The specimen consisted of a grayish-white encapsulated tumor measuring 15 by 11 by 6 cm., and a separate encapsulated tumor measuring 5 by 4 by 1.5 cm. The larger tumor weighed 473 Gm. (Figs. 5 and 6), and the smaller mass 13 Gm. (Fig. 7). The outer surface of both was smooth and glistening, with fibrous tags attached. On section the larger tumor mass (Fig. 6) varied from pale gray to light yellow, with a central portion that was quite firm and rubbery in consistency, surrounded by soft spongy tissue. The smaller tumor (Fig. 7) was pale yellow in color, and had the consistency of adipose tissue.

Microscopic: Sections of the larger tumor mass showed a malignant neoplasm composed, for the most part, of interlacing bundles and strands of spindle cells with poorly defined, slender filamentous basophilic cytoplasm and oval to spindle-shaped nuclei (Fig. 8). The latter exhibited hyperchromatism and variation in size and shape with occasional large, bizarre forms. In a few areas there were islands of adult fat cells together with less well-differentiated lipoblasts, some of which exhibited bizarre, spider nuclei with vacuolated cytoplasm (Figs. 9 and 10). The tumor presented a loose, edematous appearance in general, with areas of myxomatous degeneration and necrosis. There were foci of secondary inflammation and areas showing capillary proliferation. In some areas, bands of collagenous connective tissue were prominent. Fat stain (Sudan III) demonstrated fat vacuoles not only in the adult fat cells but also in less well-differentiated cells (Fig. 11). Bodian stains show an absence of neurofibrils.

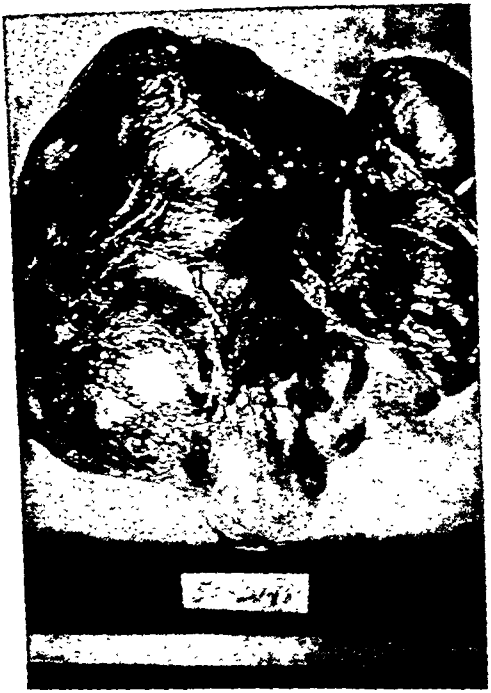

Fig. 5.—Photograph of the larger mediastinal tumor showing the encapsulated, lobulated surface.

This tumor suggested, in some areas, a fibrosarcoma with myxomatous changes. However, the presence of the adult fat cells and less well-differentiated cells taking the fat stain, and of the large, bizarre cells with spider nuclei was characteristic of a liposarcoma. The sections were reviewed by Stout,[22] who concurred in the diagnosis.

Sections of the smaller tumor mass showed adult fat cells surrounded by a fibrous capsule. Attached to the capsule was a fragment of tumor tissue containing large bizarre cells surrounded by adult fat cells presenting an appearance similar to that of the larger tumor.

The patient's postoperative course was uncomplicated except for a slight paresis of the right phrenic and recurrent laryngeal nerves, evidenced by impairment of function of the right hemidiaphragm and the right vocal cord. The motion of the right leaf of the diaphragm returned to normal within two weeks. Recovery of the right recurrent nerve occurred more slowly, but at the time of his discharge both cords moved normally and his speaking voice was better than before surgery. The patient was returned to full duty on Jan. 15, 1951, at which time his chest roentgenograms were negative except for some thickening of the right superior mediastinal and apical pleura. He was free of symptoms when discharged.

The patient returned for follow-up examination on March 9, 1951. He remained free of symptoms, his voice was normal, fluoroscopy showed no impairment of the function of his diaphragm, and his chest roentgenograms (Figs. 12 and 13) were essentially negative.

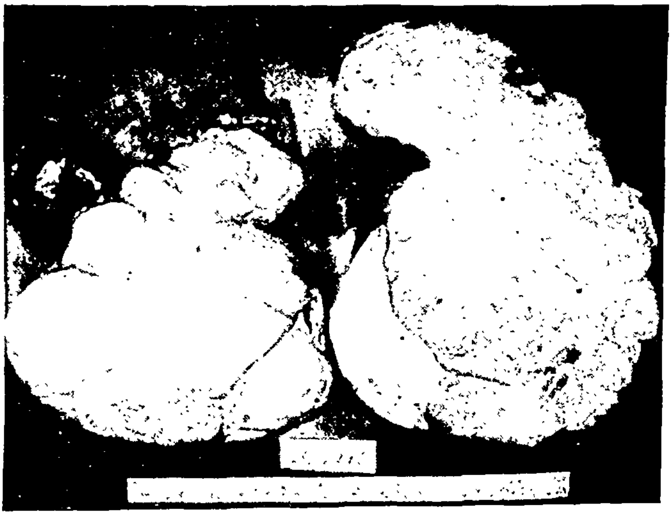

Fig. 6.—The tumor shown in Fig. 5 bisected showing the lobular pattern and variegated appearance.

DISCUSSION

Incidence.—Although Virchow[23] first recognized liposarcoma histologically in 1857, Robertson,[24] in a critical review published in 1916, was able to find only seventeen recorded acceptable cases among fifty-one fatty tumors of unusual type reported to that time. He found eleven additional cases where malignancy was questionable. Moreland and McNamara,[4] in 1942, reported nine instances of liposarcoma among a group of 16,000 patients with tumors, and suggested that the frequency of the lesion may be greater than reports in the literature would indicate. Lingley[25] reported a case in 1939 and stated that it was the seventy-second case to be recorded. In adding a case to the literature in 1949, Cunnick[26] stated that there was a total of 175 previously reported cases. Of these, the great majority primarily involved the fascial planes of the thighs or buttocks, the mesentery and omentum, or the retroperitoneal areas. Twenty-one liposarcomas were encountered in the laboratories of pathology and surgical pathology of the Presbyterian Hospital, and only forty-one such cases were accumulated in the laboratory of Surgical Pathology of Columbia University during a thirty-seven year period.[1] Considering the liposarcomas occurring in the mediastinum, such lesions appear to be extremely rare. The cases of Perkins and Bowers,[6] Ackerman and Wheeler,[2] Joske,[12] Lennert,[13] Barbier and Mollard,[14] and the one reported herein constitute the only six cases of solitary liposarcoma confined to the mediastinum of which we could find record. (See Table I.) The case of Narr and Wells[27] is not included, even though the mediastinum and entire left hemithorax were found at autopsy to be involved. Earlier films clearly indicated that the tumor arose as an encapsulated lesion from the diaphragm or chest wall at the left costo-phrenic angle. None of the forty-one cases reviewed by Stout[1] were mediastinal in location. In reporting a case showing multiple malignant fatty tumors, Ackerman[19] noted that the American literature contained no report of a similar case. He was able to find in the foreign literature, however, reports of four cases believed to represent examples of multiple primary liposarcomas.[15-18] At least two of these cases, including Ackerman's, showed mediastinal involvement.[15, 19] There is some question as to whether all of the lesions described in these reports were primary tumors or metastatic growths.

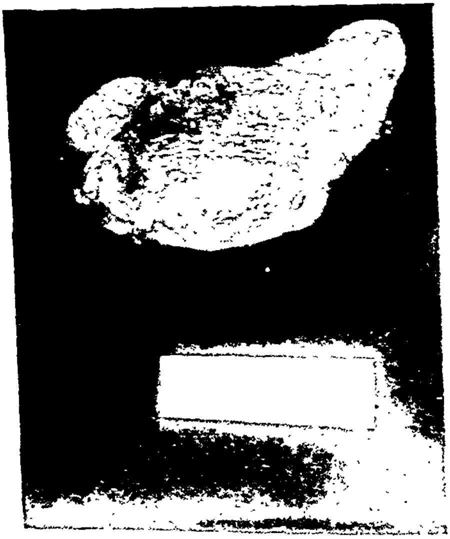

Fig. 7.—Photograph of the smaller mediastinal tumor (lipoma).

As to lipomas, mention has been made of the fact that the mediastinum constitutes a most infrequent site. Yater and Lyddane[7] reported a case in 1930, reviewed the literature, and found only eleven previously recorded cases in that

TABLE I. VERIFIED CASES OF LIPOSARCOMA OF THE MEDIASTINUM

AUTHOR	DATE	AGE	SEX	LOCATION	SIZE	METASTASES	DIAGNOSIS	TREATMENT	RESULT
Barbier & Mollard[14]	1926	25	M	Anterior mediastinum. Projected into the right hemithorax	Filled the entire right chest	None	Autopsy specimen	None	Died of mediastinal compression and respiratory embarrassment
Perkins & Bowers[6]	1939	18	F	Posterior mediastinum. Projected into the right hemithorax	Four inches in diameter	None	Surgical specimen	Surgical excision	Excellent P.O. result. Alive and well 3 yr. and 2 mo. later
Ackerman & Wheeler[2]	1942	50	F	Entire mediastinum. Projected into and completely filled left chest	Filled entire left hemithorax	None	Autopsy specimen	None	Died of respiratory embarrassment
Joske[12]	1944	18	M	Posterior mediastinum, at level of seventh rib on the left	4 × 3 × 2 in.	None	Surgical specimen	Surgical excision	Excellent P.O. result. Alive and well 1 year later
Lennert[13]	1949	23	M	Anterior mediastinum. Projected into both the right and the left hemithorax	"Size of man's head" 25 cm. in diameter	Direct invasion of right auricle	Direct aspiration biopsy. Confirmed by postmortem studies	Irradiation 4,600 r. Urethrane Cyren-A Implantation	No response to therapy. Death after 3½ mo. from respiratory insufficiency and cardiac failure
Authors'	1950	55	M	Mid and posterior mediastinum. Projected to right from superior mediastinum	15 × 11 × 6 cm. Weight 475 Gm.	None	Surgical specimen	Surgical excision	Excellent early result. Symptom free and negative chest x-rays 3 mo. P.O.

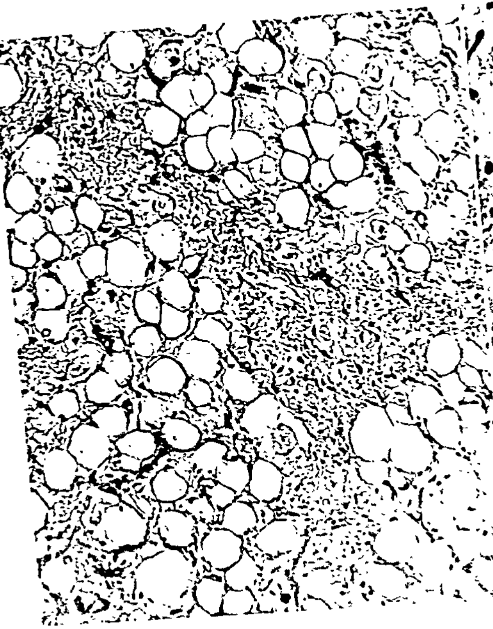

Fig. 8.

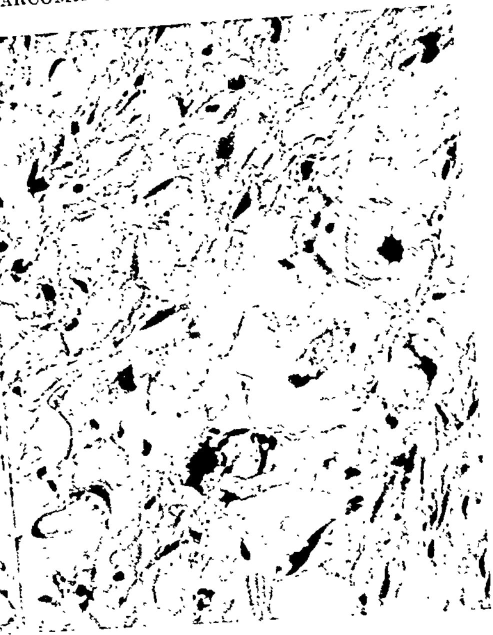

Fig. 9.

Fig. 8.—Section of the tumor showing variation from fully differentiated adult fat cells to anaplastic cellular sarcoma.

Fig. 9.—Area showing immature fat cells with a bizarre giant lipoblast with spider nucleus. There are also several spindle cells with vacuolated cytoplasm.

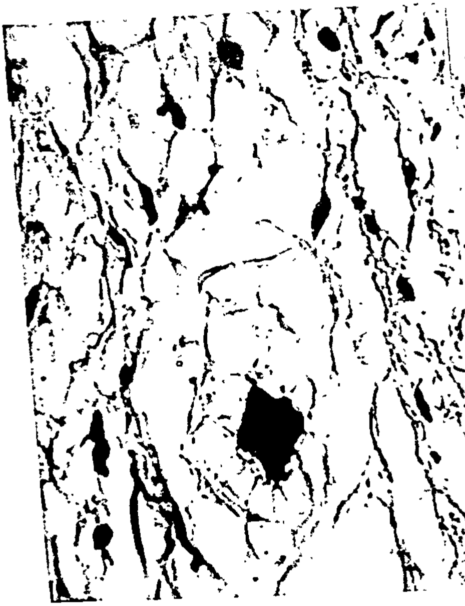

Fig. 10.

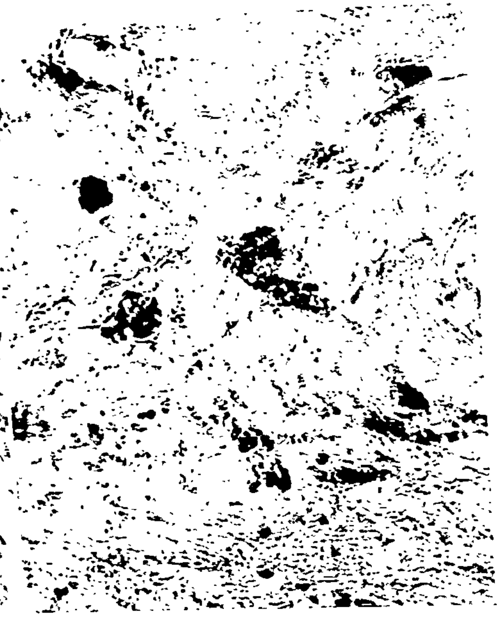

Fig. 11.

Fig. 10.—Detail of Fig. 8 showing giant "spider cell" with bizarre nucleus and vacuolated cytoplasm.

Fig. 11.—Frozen section stained with Sudan IV showing presence of fat in neoplastic cells.

location. Only one of the twelve cases was confined entirely to the mediastinum, the remainder being dumb-bell tumors with cervical or chest wall extensions. The propensity of these masses, when occurring in the mediastinum, to assume an hour-glass configuration with an extrathoracic component has been emphasized by Heuer[8] and Heuer, and Andrus and associates.[28] In 1933 Heuer[8] was able to find a total of twenty-eight cases of thoracic lipomas recorded in the world literature since the first case reported by Fothergill[29] in 1783. Of these, seventeen were confined to the mediastinum, and an additional nine cases of hour-glass tumors involved the mediastinum in part. As late as 1948, Griffin and Guilfoil[11] reported a mediastinal lipoma and stated that the total number recorded from that site still totalled less than fifty. Many of these were dumb-bell tumors and not confined exclusively to the mediastinum.

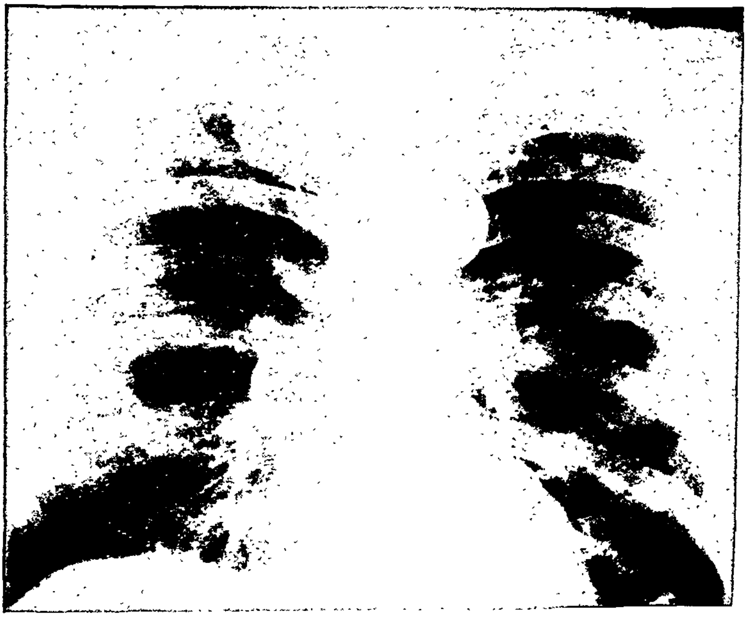

Fig. 12.—Postoperative posteroanterior and right lateral chest roentgenogram, made approximately three months after surgery, showing an essentially normal chest.

Diagnosis.—

It is not the purpose of this paper to discuss in detail the differential diagnosis of mediastinal masses nor to describe the diagnostic investigations that may be indicated in the individual case. Studies similar to those instituted in the case reported herein, including careful roentgen examination employing both roentgenography and fluoroscopy, angiocardiography[30-32] and bronchoscopy will usually be in order. Tomography will occasionally provide additional helpful information, particularly in differentiating solid from cavernous lesions. That technique will also demonstrate the presence of calcium in tumors when it may not be seen in conventional films. Roentgenograms of the esophagus with barium swallow will establish the relationship of that organ to the mass and show evidence of esophageal involvement if present. Roentgenographic studies will ordinarily offer the most assistance in arriving at a correct diagnosis. The fact that fatty tumors cast an x-ray shadow midway in density between air and water is well known. This observation led to an accurate preoperative diagnosis in the case of liposarcoma reported by Lingley.[25] Selman and Bender[33] and Hunt and Bisgard,[34] among many others, have correctly predicted the presence of a lipoma prior to surgery by that method. Even though preliminary studies fail to establish definitely the nature of the mass in question, the information gained thereby will so clearly define the exact location of the tumor and its relationship to adjacent structures that it will be most helpful to the surgeon. Despite the most painstaking investigation, thoracotomy will usually be mandatory before an accurate histologic diagnosis can be made. The principle that all unilateral circumscribed mediastinal tumors should be subjected to exploration has been widely accepted by those interested in surgery of the thorax. At operation the tumor is removed if possible, and if resection does not prove to be feasible it is biopsied in order to obtain a precise pathologic diagnosis. Knowledge that approximately 25 per cent of these tumors of the mediastinum will prove to be malignant serves to emphasize the necessity for early surgical therapy. The tendency of a significant percentage of lesions which are originally benign to undergo malignant change has been accepted by

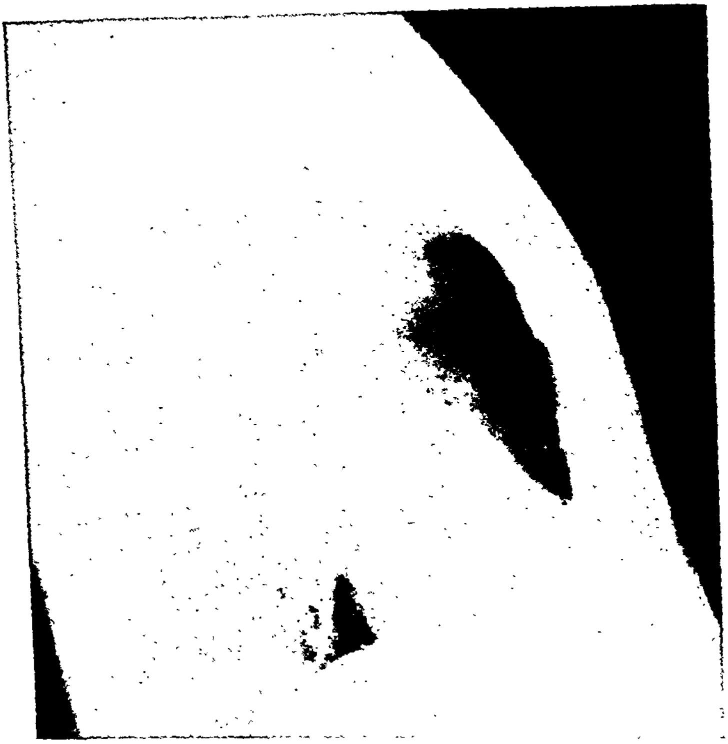

Fig. 13.—Postoperative posteroanterior and right lateral chest roentgenogram, made approximately three months after surgery, showing an essentially normal chest.

many authorities. Even those tumors which remain histologically benign can cause serious illness or even death of the patient as a result of pressure on adjacent vital structures. These facts make it abundantly clear that resection of all mediastinal tumors is indicated when excision is possible. In the final analysis, the definitive diagnosis in mediastinal liposarcomas and lipomas, as in the vast majority of mediastinal tumors, rests upon a careful pathologic examination of the entire specimen after surgical excision.

Pathology.—According to Stout,[1] liposarcomas present a variegated gross appearance. They are large, bulky, sometimes nodular, encapsulated masses ranging in color from pale yellow to orange. Areas of increased vascularity or hemorrhage may cause slight variation of this color. Most are slimy and mucoid and are surrounded by firm, fibrous tissue. Not infrequently areas of normal appearing fat are present. It is unusual to see areas of cystic degeneration. Local multiple nodules are frequent, and often account for recurrences.

Microscopic features are even more varied. Ewing[35] divided liposarcoma into two main types: the myxoliposarcoma, which is vascular, myxoid, and lipoblastic, and the granular cell lipoblastoma composed of cells resembling the foamy lipoblasts of brown fat. Stout[1] divides liposarcomas into four groups: (1) well-differentiated myxoid type, (2) poorly differentiated myxoid type, (3) round-cell or adenoid type, (4) mixed types. In the well-differentiated myxoid type, of which the case herein reported is an example, there are adult fat cells and embryonal stellate or spindle-shaped cells containing droplets of fat, which stain with Sudan III or Scharlack R. This is the less malignant group and it is questionable whether such tumors ever metastasize while they remain in this state of differentiation.

It is of special interest in this case that an encapsulated lipoma was found adjacent to the liposarcoma in the mediastinum. In addition, an encapsulated lipoma was removed from the abdominal wall which histologically showed no malignant change. Since there are several well attested cases of malignant change in pre-existing benign lipomas (Stout,[1] Schiller,[36] Adair and associates[37]) the possibility of such an origin of the mediastinal liposarcoma was entertained in the present case. However, multiple sections of the tumor failed to show definite proof of such origin. Stout[1] states that it is probable that the large majority of malignant lipoblastic tumors are malignant from their beginning. The case reported in this paper is also interesting in the light of the conception of Gideon Wells[38] that adipose tissue should be considered as a specialized organ subject to systemic disorders, similar to the endocrine glands. He noted that fat tissue, behaving as a specialized organ, could be the site of a malignant process. Siegmund,[17] in reporting a case of multiple primary liposarcomas, concluded that it represented "systemic malignant disease of fatty tissue." He compared it with lymphosarcoma and von Recklinghausen's disease. Ackerman[19] reported a similar case and reviewed the four previously described. He concluded that his case as well as the four preceding ones supported the opinion that adipose tissue should be considered as an organ subject to its own diseases. The case recorded in this report might be interpreted as adding further support to that concept.

Treatment.—The number of liposarcomas reported has been too few to enable one to formulate a rational plan of therapy on the basis of experience. Reports on the radiosensitivity of these tumors vary greatly. Ewing[35] stated that many of them show considerable radiosensitivity, and he suggested that radiation therapy be used with surgical excision of the residual tumor if demanded. Geschickter[3] feels that these tumors are radiosensitive, but he does not claim that they are cured by that method. He found ultimate recurrence and metastasis to be the rule. Review of the reported cases leads one to agree with Ackerman and Wheeler[2] that the value of irradiation is questionable, though some tumors have been proved to be definitely radiosensitive.[1, 3] The differing opinions which have been expressed concerning the necessity for radical surgery are probably a reflection of the varied pathologic characteristics which liposarcomas present. Thus Goormatigh and associates[18] believe that the name liposarcoma has a more ominous significance for the surgeon than is warranted by the slow growth of the tumor. Ackerman and Wheeler[2] observe that the degree of malignancy is not considered as high as that of other sarcomas. With reference to liposarcomas of the breast, de Navasquez and Horton[39] have expressed the opinion that radical surgery is not necessary for long survival. On the other hand, Starkloff and associates[21] and Morgan[40] have recommended amputation when the lesion is situated in a location where such a procedure can be considered. McCormick[41] advises preoperative irradiation and radical surgery. Ackerman and Wheeler[2] propose removal with postoperative irradiation if there is doubt as to complete excision. They also suggest that irradiation be tried in inoperable cases. The most rational therapeutic program seems to be that outlined by Stout,[1] who bases his advice on the relative degree of malignancy of the lesion. In the well-differentiated tumors he feels that generous local excision is sufficient, for the worst that may happen in this group is local recurrence. In all other liposarcomas, consisting as they do of more highly malignant tissues, radical surgery is urged. Stout[1] does not feel that irradiation is a form of therapy which should be entirely abandoned, and the circumstances of an individual case may dictate its use. Insofar as mediastinal liposarcomas *per se* are concerned, the deep-seated location of these tumors makes surgical excision even more urgently indicated than in the superficially situated lesions. In attacking these growths surgically the greatest care should be exercised to remove the entire capsule and the tissue immediately adjacent to it in order to assure the most complete possible resection of all malignant elements.[1]

Prognosis.—Patients with liposarcoma must be offered a guarded prognosis. Those with a well-differentiated tumor have a favorable outlook as long as the lesion retains its differentiation.[1, 39, 42] The infiltrative nature of these neoplasms is shown by their ability to recur locally on repeated occasions, sometimes after a long free interval.[1-3, 25, 35, 41-44] Metastases are the rule in the less differentiated types.[1, 2, 3, 25, 41, 44] They may be by way of either the blood or lymph streams and occur most commonly to the liver and lungs. Metastatic lesions may result in death of the patient by invasion of a vital structure or from secondary pressure effects. McCormick[41] has emphasized that one may be deluded into offering a good prognosis in a case of liposarcoma because of its apparent encapsulation when such an optimistic outlook is not justified in view of the nature of the neoplasm.

SUMMARY

A case of primary liposarcoma of the mediastinum associated with a mediastinal lipoma and a subcutaneous lipoma of the anterior abdominal wall is reported. This appears to be the sixth case of solitary mediastinal liposarcoma to be recorded in the literature. There are only three reported instances of the concomitant occurrence of liposarcoma and lipoma, and there is no record of the simultaneous occurrence of mediastinal liposarcoma and lipoma.

The mediastinal tumors were successfully resected, making this the third recorded mediastinal liposarcoma to be removed surgically. We believe that surgical excision is the treatment of choice.

The literature pertaining to liposarcoma is briefly reviewed.

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A DEVICE FOR PRODUCING REALISTIC PULSATION OF A DEAD HEART

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NEW operative techniques must be worked out as fully as possible on the cadaver and experimental animal before they can be applied to the human being. This is especially true in cardiac surgery where success depends on rapid faultless procedure, and there is no margin for clumsiness or error. The pioneers of heart valve surgery have very properly recorded cases in which death was directly attributable to technical failures.[1, 2]

Experimental operations on animals are invaluable, but not entirely adequate, since very large dogs are difficult to obtain, the anatomy not identical, and cases of stenotic valvular disease never found. Operations on the cadaver allow study of the anatomic approach, but give no information on such vital matters as the control of hemorrhage or the feel and handling of pulsating structures.

To overcome this difficulty, a simple apparatus has been designed which when filled with citrated blood and attached to the dead heart causes that organ to pulsate in a most realistic manner. It can be used in vitro or in situ with a minimum of disturbance to the thoracic viscera, and is easily and rapidly connected. The rate and volume of the pulsations may be altered at will.

The close similarity of the contractions and valve movements thus produced in dead dogs' hearts to those of the living animal has been amply confirmed. The findings on digital palpation in a case of mitral stenosis tallied closely with those found at operation by Brock.[3]

The procedure has so far been used mainly for experimental mitral valvulotomy in the dead human heart.

CONSTRUCTION

The apparatus (Fig. 1) consists essentially of two rubber bulbs of 250 c.c. capacity each, enclosed in an airtight glass fronted box. This latter is connected to a pulsator which alternately compresses and expands the air around the bulbs and thus indirectly their contents.

The bulbs are connected by short wide bore rubber tubes to cannulae tied into the aorta and pulmonary artery, respectively. Manometers are fitted and pressure bottles enable any lost blood to be replaced with the apparatus still in action. For the sake of clarity only one pressure bottle and cannula setup is shown.

METHOD OF SETTING UP THE APPARATUS

The following arrangement has proved suitable for experimental mitral valvulotomy. With a little ingenuity it may be adapted for any cardiac operation.

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The aorta is cut off within 2 in. of its origin, and its valve cusps slit transversely with Mayo scissors to produce complete incompetence. The pulmonary veins are then ligated, and the cannula tied into the aortic stump. The clip *A* is then opened, and the left heart and apparatus slowly filled with blood from the pressure bottle; the ventricle being squeezed all the while to get rid of any surplus air. The clip *A* is then closed, and any remaining air bubbles expelled through the side tube *B*. It will be found that the auricle slowly fills with blood even though the valves are normally competent. A similar procedure is carried out with the pulmonary artery after the venae cavae have been ligated.

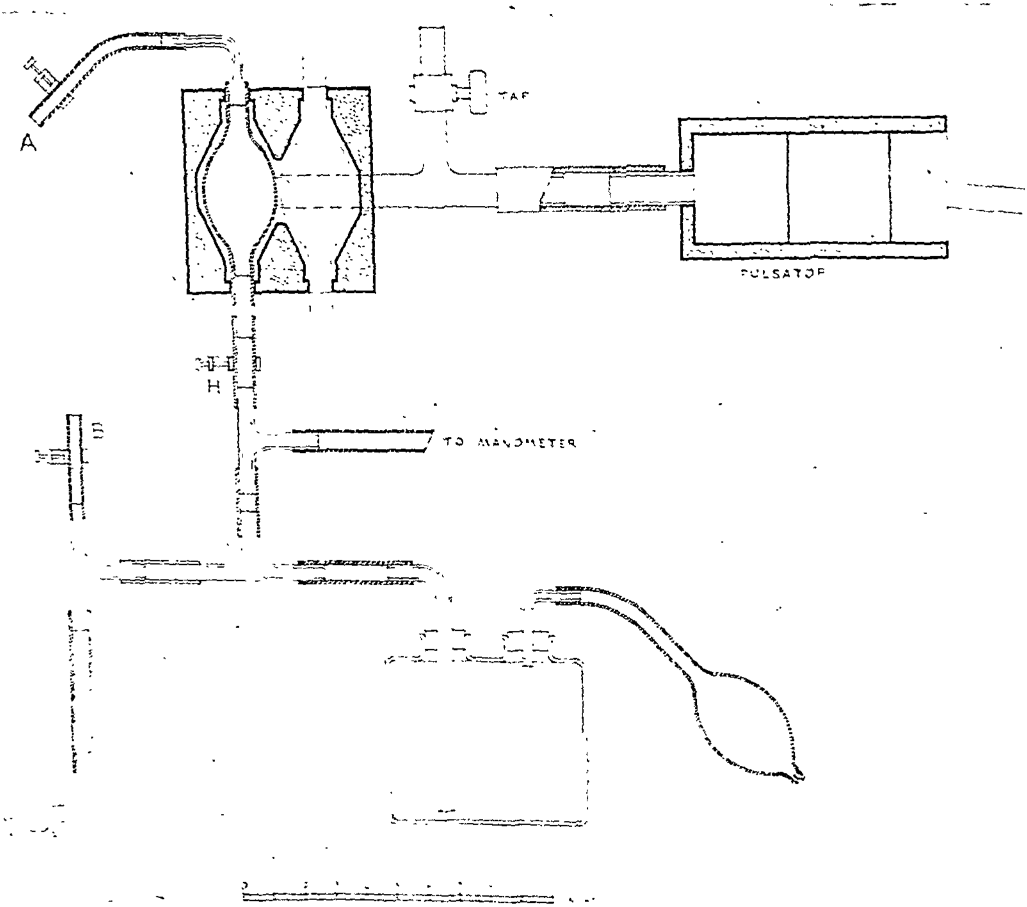

Fig. 1.

The pulsator is then started and the heart beats in a most realistic manner for as long as may be required. The pressures in either side of the heart can be measured with the manometers, and adjusted as desired by means of the screw clamps *H* and *H′*.

Should operative procedures result in serious "hemorrhage," blood may be replaced in known quantities from the pressure bottles without interfering with the heart in any way.

When operations are carried out on the mitral valve with the heart in situ the aorta may be conveniently reached by resecting the first 3 in. of the right *second costal cartilage and rib. An extension downward through the third* costal cartilage will bring the pulmonary artery within view. The left chest is thus left virgin for surgical procedures.

DISCUSSION

With every new operation the mortality declines as experience is gained. Anything that will reduce the number of these early disasters and enable the inevitable mistakes to be made on the dead rather than the living is worth while. The *artificially pulsating heart may therefore come to have a definite place* in the development of new procedures, and in the training of cardiac surgeons. With it intracardiac structures may be examined, and procedures carried out on pathologic hearts under realistic conditions. As in no other way skill may be acquired in cutting rapidly moving stenosed valve rings. Serious mistakes while entering or leaving the heart cavities will result in torrential "hemorrhages" exactly simulating the consequences of mishandling in the living. Also emergencies, e.g., the tearing out of the purse-string suture around the base of the auricular appendage, may be deliberately produced to give practice in dealing with potential disasters.

Cardiac surgery units will find this simple apparatus well worth a trial.

SUMMARY

1. The necessity for the utmost surgical skill in cardiac operations is strongly emphasized.

2. Reasons are given why completely adequate experience cannot be gained on animal or cadaver hearts.

3. A new apparatus is described for producing realistic pulsations of the dead human heart, thus making possible experimental operations under almost natural conditions.

4. A method of setting up the apparatus for mitral valvulotomy is given.

5. Some uses for the apparatus are discussed.

Acknowledgement is made to Prof. R. D. Wright, Professor of Physiology in the University of Melbourne, both for the provision of experimental facilities and for constant help and encouragement.

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A NEW OPERATING CARDIOSCOPE

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THE value of blind commisuriotomy in certain types of mitral stenosis is becoming widely recognized,[1-3] but until the valve orifice can be inspected, and the commisures cut under direct vision, accidents would seem inevitable.[4] Cardioscopes of the traditional lens-in-tube construction have not proved entirely satisfactory. A new instrument has therefore been devised, which, while retaining the contact vision principle, utilizes the unique light transmitting properties of solid perspex (methyl methacrylate). A wide brilliantly illuminated field is obtained through which intracardiac structures can be clearly seen in the living animal.

This cardioscope (Fig. 1) can be made within a few hours in any laboratory workshop. It has the following important advantages over previous types. (1) Simplicity: the entire instrument, apart from the operating knife, is formed from solid perspex of uniform refractive index. (2) The visual field extends to the extreme edge of the instrument without obstruction by light bulb or metal casing. (3) There are no lenses to become misted over or produce aberration. (4) The light source is outside the instrument, it can therefore be really adequate, and if necessary replaced in a few seconds with the instrument still in situ. (5) The tip may be fashioned to any desired plane surface or angle (Fig. 3) without distortion or light loss. (6) Polished perspex is easily sterilized by immersion in cetyl trimethyl ammonium bromide, it does not transmit heat, and its water-repellent surface is ideal for contact with delicate tissues.

CONSTRUCTION

The instrument consists of a perspex body containing the cutting mechanism, with a total weight of 6 oz., and a detachable light holder. The whole can be rapidly taken apart for cleaning.

The body 10½ in. long and 1 in. in diameter is made from solid perspex rod turned down at one end to form a 2½ in. operating shaft ½ in. in diameter, which is bored to carry a 5/32 in. knife and haft. Both ends are very carefully polished to a dead flat transparent surface. The light transmitting arm also of 1 in. diameter rod is cemented to the main body with perspex solution to form a single homogenous unit.

The knife haft passes through the operating shaft and along a groove in the body of the instrument to be attached to a spring loaded plunger. This arrangement insures that the knife is automatically withdrawn into the operating shaft when not in use.

Various types of shaft tip and operating knife that have been used are shown (Figs. 3 and 4). The hooked knife (Fig. 4 C) was found especially

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useful in cutting the mitral valve cusps of normal animals; unlike the others, however, it has the disadvantage of not withdrawing completely into the shaft. Blunt headed knives are less liable to cause injury, but, unless double edged, necessitate the cardioscope being turned through 180 degrees to cut both ends of a stenosed mitral orifice.

The source of light, a 24 watt, 12 volt bulb, is carried in a jacketed holder through which water circulates continuously. It is secured to the light transmitting arm by a single screw.

EXPERIMENTAL USE

The cardioscope has been tested mainly on the mitral valves of dogs, being introduced via the left auricular appendage using Brock's purse-string technique.[5] A very satisfactory view of the valve cusps, chordae tendineae, and mitral ring was obtained in every case, and it was possible to cut the valve cusps at any desired point.

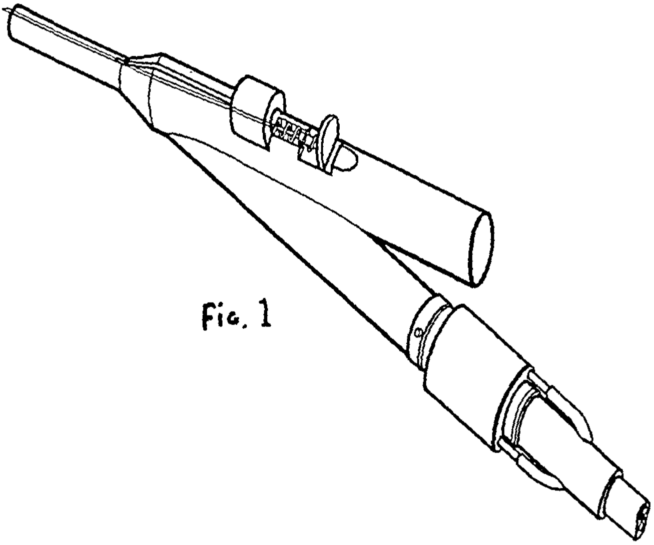

Fig. 1

The instrument was well tolerated within the auricle for upwards of one hour. In one dog, a greyhound, it was passed right through into the ventricle and the papillary muscles inspected without causing undue disturbance. Since the instrument never entirely blocked the mitral orifice there was no violent reaction like that following the insertion of the index finger.[6] Dogs whose posterolateral valve cusps had been cut under direct vision made an uninterrupted recovery.

Operations on the blood filled cadaver heart showed that a stenotic valve could be clearly visualized and accurately cut along the commisures as far as the mitral ring.

In the later experiments a cardioscope with an angulated tip was used. This allowed operation to be carried out through a much smaller thoracic incision without any loss of visual acuity.

DISCUSSION

This instrument allows operations on the mitral valve to be carried out under direct vision. It should therefore be especially useful when the valve cusps are grossly distorted or calcified. In these cases blind maneuvers sometimes split the valve at its weakest point, i.e., transversely, instead of along the commisures, with fatal results.[7] As with other endoscopic procedures, skill comes rapidly with practice and it should be possible to cut a stenosed valve at any point desired without risk of damaging the chordae tendineae.

Every extra maneuver inside an enfeebled heart must increase the risk of a fatal arrythmia; with this instrument the valve can be inspected and cut in one operation. It should be possible to deal adequately with a stenosed valve in less than 60 seconds.

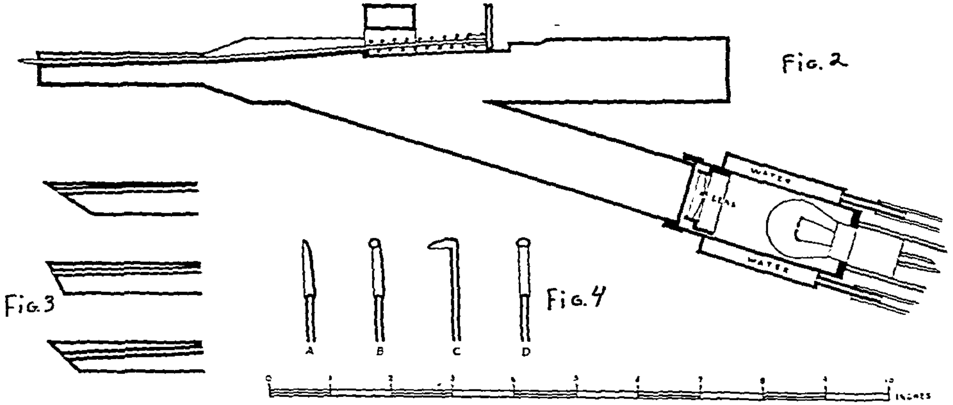

Fig. 2

Fig. 3

Fig. 4

The operating shaft being much thinner and more easily introduced than the index finger (diameters as little as one-third inch give a fair field of vision), other uses suggest themselves. For example, a modified form of instrument might be introduced via the innominate artery in cases of aortic stenosis without undue interference with the cerebral blood supply. Further work is proceeding on these problems.

SUMMARY

1. The necessity for an efficient cardioscope is discussed.

2. The advantages of utilizing the unique properties of perspex are enumerated.

3. Constructional details of the new instrument are given.

4. Some experiences with the mitral valves in living dogs and the human cadaver are described.

5. Certain types of stenosis where operations under direct vision seem especially necessary are referred to, and speculations made as to other possible procedures.

Acknowledgement is made to Prof. R. D. Wright, Professor of Physiology in the University of Melbourne, both for the provision of experimental facilities and for constant help and encouragement.

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NOTICE OF ANNUAL MEETING

The American Association for Thoracic Surgery

The Thirty-second Annual Meeting of the American Association for Thoracic Surgery will be held May 8, 9, and 10, 1952, in Dallas, Texas. Headquarters will be at the Hotel Baker. The Adolphus Hotel and the Southland Hotel are near by and will also have suitable accommodations.

Requests for Hotel Reservations: These should be mailed directly to S. Hugh White, Room Reservation Manager, Hotel Baker, Dallas 1, Texas.

Abstracts for Papers: Abstracts for the presentation of papers at the 1952 Meeting must be received for consideration by the Program Committee on or before Jan. 1, 1952. These abstracts should be submitted in *quadruplicate* and addressed to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor. Abstracts received after Jan. 1, 1952, will not be considered.

Applications for Membership: All correspondence regarding applications for membership, *including* requests for membership applications should be addressed to the Chairman of the Membership Committee, Dr. Edward M. Kent, 1107 Clark Bldg., Pittsburgh 22, Pa. Completed membership applications must reach Dr. Kent on or before Jan. 1, 1952, in order to be acted upon at the annual meeting in Dallas.

PRIMARY LYMPHOMA OF THE LUNG

Review of the Literature, Report of One Case, and Addition of Eight Other Cases

William C. Beck, M.D., and John C. Reganis, M.D.
Sayre, Pa.

Primary lymphoma of the lung is a rare disease. Some case reports are to be found in series of various types of lymphoma, as Sugarbaker and Craver,[1] who found one instance of a primary pulmonary tumor in a series of 196 lymphosarcomas. Others may be found in some of the series of solitary lung tumors, as in Thornton, Adams, and Bloch's[2] report of twenty-three cases of solitary lung tumor, of which one was listed as a lymphoma. Only six reports, however, have been found in the literature which specifically recount individual cases of primary pulmonary lymphoma.

To this group of six individual case reports we would like to add one which we have studied. To add further to the knowledge of the incidence, the behavior, and the therapeutic possibilities in dealing with this tumor, we wrote to fifteen surgeons who have had wide experience with thoracic tumors. Of these, only five reported that they had seen cases of primary lymphoma of the lung. These five surgeons have observed a total of eight such patients, and they have given us some of the information concerning their results in the care of these.

While the number of cases, both in the literature and those gleaned from the reports furnished by these surgeons, is small, certain conclusions may be drawn regarding this rare tumor. The picture which may be drawn from this meager information will probably require amendment as more cases are found through mass radiographic studies. An analysis of the information available of the fifteen cases of which we have knowledge is to be found in Table I.

Our interest in this subject was stimulated by the following case.

CASE REPORT

A 47-year-old housewife was admitted to the Guthrie Clinic Nov. 8, 1949, complaining of a dull aching pain in the epigastrium, present for two months. The pain was aggravated by most foods, especially fatty and leguminous ones. There was neither nausea nor vomiting. She had a 10 pound weight loss in the previous two months. Some relief was experienced with antacid proprietary drugs. The referring physician felt that clinically she had a peptic ulcer.

Systemic and family review were unrevealing. There were absolutely no pulmonary symptoms. The physical examination revealed a moderately obese woman, with all physical signs negative except mild tenderness in the epigastrium. Extensive laboratory studies revealed only a mild microcytic anemia. The study of the gastric acidity by the caffeine method revealed a slight elevation. The blood sedmentation rate was normal. The routine chest film (Fig. 1) showed a discrete, smoothly rounded shadow in the lower lobe of the right lung near the costophrenic angle about 6 cm. in diameter. The remainder of the lung fields were clear, and the mediastinal shadow was normal. The radiologic examination of the stomach revealed a constant defect along the greater curvature of the stomach with a crater superimposed on it.

It appeared most likely that this patient was suffering from a gastric carcinoma with metastasis to the chest. Therefore, gastroscopic study with the operating gastroscope was carried out. This revealed what was believed to be a healing gastric ulcer with a granulating

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From the Surgical Service of the Guthrie Clinic.

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TABLE I.

AUTHOR OR SURGEON	AGE	SEX	SITE	SIZE	SYMPTOMS	METASTASES	BRONCHOSCOPY	OPERATION	RESULT
Pekelis[4]	–	–	L.L.L.	"Orange"	No	Yes	No	No	
Spatt & Grayzel[5]	34	F	R.L.L.	17 by 11 cm.	Yes	No	Yes	Lobectomy	alive 14 mo.
Churchill[6]	45	F	R.U.L.	15 by 11 by 7 cm.	No	No	Yes	Lobectomy	well 6 mo.
Maier[3]	50	M	L.U.L.	massive	No	No	Yes	Pneumonectomy	well 19 mo.
Weissman & Christie[7]	52	M	L.U.L.	massive	Yes	No	Yes	Pneumonectomy	well 15 mo.
Anlyan, Livingood & Klassen[8]	63	M	R.L.L.	6 cm.	No	No	Yes	Lobectomy	well 15 mo.
Beck and Reganis	47	F	R.L.L.	6 cm.	No	No	No	Lobectomy	well 15 mo.
Behrend*	59	F	L.L.L.	—.	Yes	No	Yes	Pneumonectomy	well 13 mo.
Touroff*	–	–	R.U.L.	5 cm.	–	No	–	Lobectomy	well 30 mo.
Betts & Overholt*	–	M	R.U.L.	massive	–	Yes	–	Inoperable	died 3 mo.
Blades*	–	M	–	massive	–	Yes	Yes†	Inoperable	died 9 mo.
Ochsner & deCamp*	66	M	–	—	–	Yes	–	Autopsy	
	–	M	L.U.L.	—	–	No	–	Lobectomy	died 4 yr.
	50	F	L.U.L.	—	–	No	–	Pneumonectomy	well 9 yr.
	46	M	R.U.L.	—	–	No	–	Pneumonectomy	well 19 mo.

*The cases reported above from the services of Dr. Hehrend, Dr. Arthur Touroff, Drs. Richard Overholt and Reeve Betts, Dr. Brian Blades, and Drs. Alton Ochsner and deCamp, were personal communications to the authors from approximately fifteen clinics polled.

†This is the only bronchoscopic study that yielded a positive biopsy.

base. The remainder of the stomach appeared to be normal. Biopsies were taken through the gastroscope.

The pathologist studied nineteen separate sections of tissue. He found the gastric epithelium to be completely benign with preservation of the normal architecture. The ulcer showed the typical degeneration found in the base of a gastric ulcer with necrosis and some inflammation, but no evidence of malignant cellular activity.

After considerable consultation and discussion between the gastroenterologist, the pathologist, and the surgeon, it was decided that an exploratory thoracotomy was indicated.

Thoracotomy was performed through the bed of the seventh rib. The pleural cavity was found to be entirely free. There was a firm mass palpable in the lower lobe of the right lung. There was no demonstrable enlargement of the hilar lymphatics and the mediastinal lymphatics appeared to be entirely normal. The lower lobe of the lung was resected, in the usual manner.

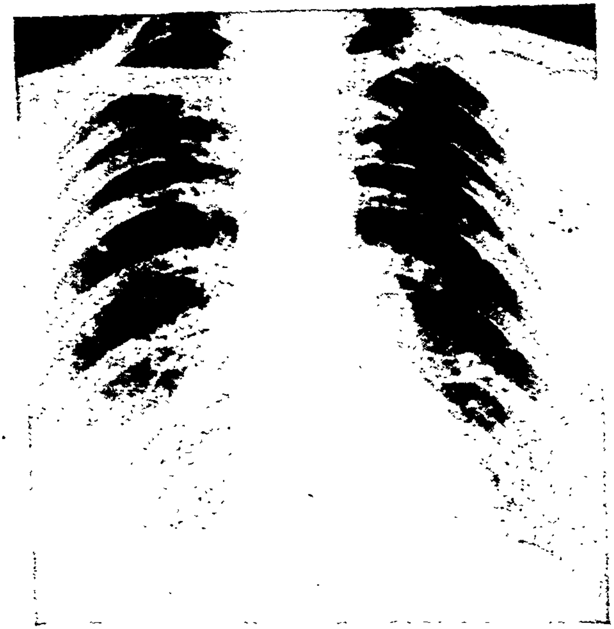

Fig. 1.—Discrete, smoothly rounded shadow in right lower lobe near the costophrenic angle.

The gross description was a mass, roughly globular measuring about 8 cm. in diameter (Fig. 2). Surrounding the mass there was normal lung tissue, containing air. Peripheral to the tumor there was some atelectatic lung tissue. Microscopically (Fig. 3) the lung tissue surrounding the tumor appeared approximately normal. The tumor itself was made up of lymphoid tissue, consisting of small round cells having a complete absence of stroma with exception of some remaining strands of pulmonary tissue. These cells invaded the pleura where the tumor was in contact with it, and extended into the alveoli of the subadjacent lung. There were small groups of cells simulating *follicles*, but actually circumscribing blood vessels. The diagnosis of lymphosarcoma (Dr. C. H. DeWan) was confirmed in the Armed Forces Institute of Pathology.

During convalescence from the operative procedure, the patient was placed upon an ulcer regimen, and her gastrointestinal symptoms disappeared. Before she was discharged from the hospital, a gastrointestinal roentgenologic study was performed and revealed complete healing of the ulcer. She had been followed on an ambulatory basis with repeated chest films and studies of the stomach and duodenum over a period of fifteen months. She has been in splendid health during this time without any evidences of either pulmonary or gastrointestinal disease.

Discussion.—

The adventitious finding of a pulmonary tumor was the result of the routine study of the chest which is made on all of our patients. The attempt to correlate all findings into one disease entity might well have resulted in abandoning this patient as hopeless, with pulmonary metastasis from a gastric cancer. The reliance placed upon the gastroscopic biopsy certainly was proved warranted.

It is interesting to note that the patient reported by Herbert C. Maier[3] was also examined because of a chronic gastric ulcer. This is, in our opinion, coincidental, but indicates the value of complete examination of every patient. It also emphasizes the value of the exploration in all solitary tumors of the lung.

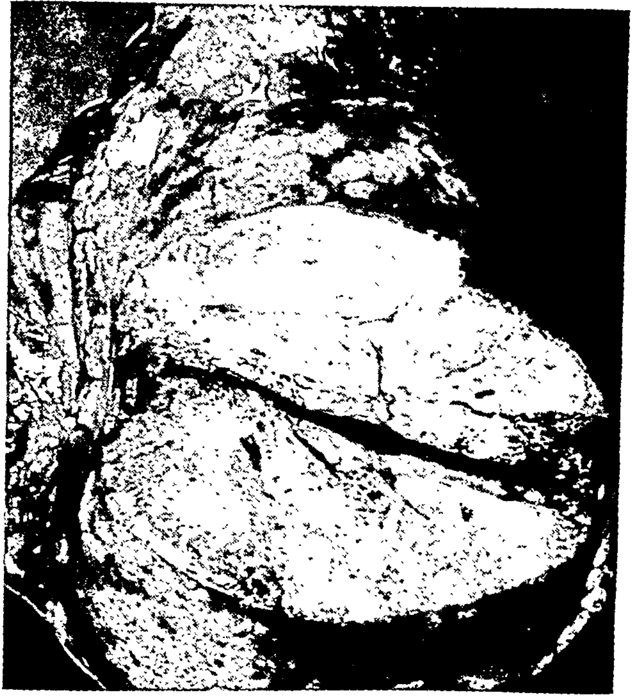

Fig. 2.—Gross cut section of lymphoma within the lung showing the typical hyaline appearance of lymphosarcoma.

DISCUSSION

In this review there were available for study fifteen cases. Six of these cases are derived from articles in the literature, eight from reports sent to us by surgeons but as yet unpublished, and one from our own service. Only one of these fifteen is an autopsy report, the others all having been studied, diagnosed, and treated during life. In seven we have the clinical background; in these only three had symptoms referable to the tumor. The other four were diagnosed only after the tumor was observed in a routine roentgenogram study of the chest. The three with symptoms all had cough varying from three months to one year. Two of these also had hemoptysis. In one patient there was also extensive weight loss, weakness, and lassitude. This patient had an extensive involvement of the entire upper lobe. Nevertheless, this patient survived a pneumonectomy for at least one year.

The sex incidence showed a moderate preponderance in males. This is typical of both lymphosarcoma and lung cancer of other types. The ages varied from 34 to 66 years, which also is typical for both lung cancer and for lymphosarcoma.

Cytologic studies of the sputum were made in four cases, but in none was it positive. Bronchoscopy was carried out in six of the fifteen patients. In three it was reported to be normal. In one it revealed suppuration in the affected lobe. In one it showed a narrowing and distortion of the bronchus in the affected area. In only one case was the tumor revealed and a positive biopsy taken. This was, of course, the only patient in whom a preoperative diagnosis was made, or even suspected. Unfortunately it was also not resectable.

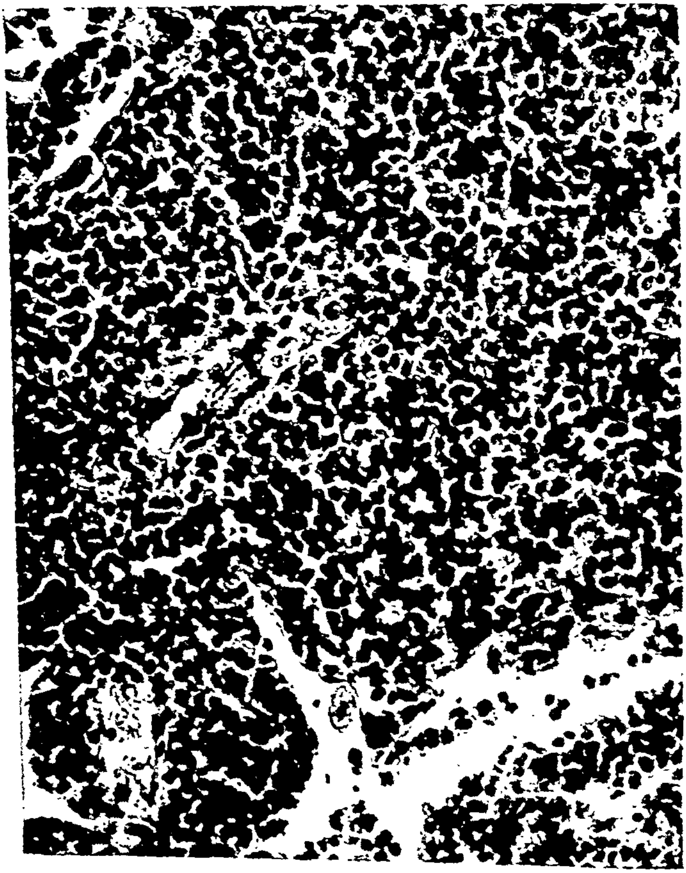

Fig. 3.—High magnification photomicrograph of lung tumor showing lymphomatous elements of fairly uniform size replacing the usual lung architecture.

The size of the tumors varied greatly. The smallest was reported as being 5 cm. in diameter. Several were massive, filling an entire lobe of the lung. Most of the tumors were discrete and, although they showed some local infiltration and were devoid of a capsule, they were definitely demarcated from the normal lung. Only four of the fifteen cases had metastases at the time of the operation. The mediastinum was involved in all of these; the liver, ribs, sternum, and brain being the sites of the more distant metastatic processes.

Of the fifteen patients in this group, thirteen were subjected to exploratory thoracotomy, and in eleven of these resective surgery was possible. Six were treated by lobectomy, while in five a pneumonectomy was performed. There was no surgical mortality in the group. We do not have late follow-up studies on any of the patients. The two patients who were explored but where the lesion was not resectable died at three and at nine months following the intervention. In both of these radiation therapy was used but appeared to be ineffectual. One patient died of a local recurrence and mediastinal extension four years after lobectomy. Of the ten remaining patients, we have some information in nine. All appeared to be in good health from six to thirty months following the operation. One patient (Ochsner and deCamp) was alive and well nine years following a pneumonectomy.

SUMMARY AND CONCLUSIONS

Primary lymphoma or lymphosarcoma of the lung is a rare disease, but should be found with increasing frequency with the more widespread use of mass radiography.

The six cases which have been individually reported in the literature have been analyzed. To this we have added one case of our own and eight others which were described personally to us by other surgeons. All of these have been reviewed from the standpoint of symptomatology, diagnosis, and therapy.

The diagnosis of pulmonary lymphoma can be made only after the tumor has been surgically explored.

The therapy appears to be resective surgery. It is impossible to state from this review whether there is any advantage in pneumonectomy over lobectomy. It appears, however, that lobectomy is probably adequate if the lesion is peripheral and small.

It is impossible to evaluate the results of radiation therapy in this disease. It would appear at least that its place is certainly secondary to resective surgery.

The importance of the surgical exploration of solitary lung tumors is stressed, even when it appears that these may be secondary to another focus.

We would like to express our appreciation to Drs. Moses Behrend, Arthur Touroff, Richard Overholt, Reeve Betts, Brian Blades, Alton Ochsner, and Paul T. deCamp for their kindness in furnishing information of their cases.

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Original Communications

RESULTS IN 278 PATIENTS WHO HAD THE MODERN TYPE OF THORACOPLASTY FOR TUBERCULOSIS

Follow-up of Living Patients Five to Fifteen Years After Operation

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THE scarcity of information about the long-term results obtained from thoracoplasty in pulmonary tuberculosis prompted us to undertake this study.[1] Reports from different clinics have appeared recently with increasing frequency, but few, if any, are based upon follow-up examinations made five years or more after the performance of the so-called modern type of thoracoplasty (as described by Alexander[2]).

Because of the great difficulties that would be encountered in following patients after their return to many different sanatoria, it was decided to study only those patients who had been referred to the University Hospital by the Michigan State Sanatorium, at Howell, Mich., and to which almost all of them returned for their convalescence.

The use of patients only from the Michigan State Sanatorium permitted us to study a relatively large group of persons who were followed actively by the Outpatient Department and all of whom had cultures made of sputum or gastric contents. All the patients were transferred to the University Hospital for operation and all the thoracoplasties were performed between Jan. 1, 1935, and Jan. 1, 1945. January, 1935, was chosen as the beginning date for this study as the modern type of thoracoplasty had become routine by that time; January, 1945, was selected as the terminal date as a minimum five-year follow-up could thereby be secured, since the study was completed at the end of 1949.

Three hundred seventeen patients from the Michigan State Sanatorium had a total of 1,388 stages of thoracoplasty performed by various members of the thoracic surgery staff of the University Hospital. All patients who were

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not known to be dead were sent questionnaires in order to establish contact with them. Most of the patients returned to the sanatorium for a check-up examination. Some did not return due to the long travel time required by the location of their homes, or were unwilling to come. For this group, we arranged to have a roentgenogram made near their homes and sent to us. Also, one or more sputum specimens were collected and sent either to the Michigan State Sanatorium or to a near-by state laboratory for culture, and the reports were then sent to us. If any patient had had a positive sputum culture at any time after discharge from the sanatorium, three specimens were required from him before he was considered negative.

Thirty-nine patients did not answer the questionnaire, and no information about them could be obtained through local health authorities, local chapters of the National Tuberculosis Association, or the police. Since follow-up data of more than five years were not available about them, they have been excluded from the study, leaving 278 patients for analysis. We feel that this is reasonable, since a study of the case records strongly suggests that the majority of these thirty-nine patients were progressing favorably. Thirteen patients of this group were followed for less than one year after operation, and only one remained positive (three had negative cultures, and nine had negative concentrates) thirteen were followed for one to two years (one remained positive, two had negative cultures, and ten had negative concentrates); ten were followed two to three years (one remained positive, six had negative cultures, and three had negative concentrates); three were followed for three to five years (three had negative concentrates); the patients mentioned above as having negative concentrates had no cultures made since the study of their sputa was prior to 1940 when routine cultures were begun. A number of them were migrant Mexicans and Indians, and eight others had moved to other states. Therefore, to list the thirty-nine as dead would, we feel, give a falsely unfavorable impression of the value of thoracoplasty. Nineteen other patients failed to answer the questionnaire, but are included in this study because we had adequate data about them five years or more after operation.

Of the 278 patients in this study there were 145 men (52.2 per cent) and 134 women (47.8 per cent). The youngest patient was 12 years old and she is now well, has a moderate scoliosis, is married, and has borne children. The oldest patient, now dead, was 64 years old at the time of operation. Table I gives more details concerning the age distribution.

TABLE I. AGE GROUPS BY DECADES

Age	10-19	20-29	30-39	40-49	50-59	64
Number of Patients	21	106	84	48	18	1
% of Total	7.5	38.1	30.2	17.3	6.5	0.4

The majority of the patients were white (258, or 92.8 per cent). There were nine Negroes (3.2 per cent) and eleven Mexicans or Indians (4.0 per cent). Two hundred thirty of the patients (82.7 per cent) had bilateral disease. Among these, the lesion in the contralateral lung was apparently active in 115

(41.4 per cent), and in three of the 115 the lesion was obviously progressive. Fifteen of the 230 had cavities visible in the so-called "better lung." Unilateral disease was present in forty-eight; twenty-two in the right and twenty-six on the left. Operation was performed on the right in 132 patients, and on the left in 146.

Among the 278 patients, positive sputum was present in 263 (94.6 per cent). Fifteen patients (5.4 per cent) had negative sputa, even though eleven of these had visible cavities by roentgenogram. The thoracoplasties of these fifteen patients were performed before 1940, and so cultures were made in only eight instances; in three only smears were used, and in four concentration of the sputa was utilized.

The amount of sputum raised by the patients varied considerably. In general, it may be stated that 157 patients (56.5 per cent) brought up less than 25 Gm. daily and thirty-one patients (11.1 per cent) raised more than 100 Gm.

One hundred eighty-nine patients (67.9 per cent) had been in the sanatorium for two years or less when their thoracoplasty was performed. Eighteen patients (6.5 per cent) were treated for three months before thoracoplasty, 36 patients (12.9 per cent) for three to six months; 75 patients (26.9 per cent) for six to twelve months; 60 patients (21.6 per cent) for one to two years; 29 patients (10.4 per cent) for two to three years; 26 patients (9.3 per cent) for three to four years; 18 patients (6.5 per cent) for four to six years; 7 patients (2.5 per cent) for six to eight years; 9 patients (3.2 per cent) for more than eight years. During this time, these patients had had, in addition to bed rest, pneumothorax (with or without pneumonolysis) or a temporary phrenic nerve interruption in a large majority of instances. Pneumoperitoneum was not used in any patient. Streptomycin, penicillin, or other antibiotic substance was not utilized either before, during or after operation.

On the side of the thoracoplasty 57 patients (20.5 per cent) had moderately advanced tuberculosis, and 221 (79.5 per cent) had far-advanced lesions. Four had exudative lesions and 87 were of the pneumonic type, while 186 were of the productive type. One patient could not be classified. The disease was limited to the apical portion of the lung in three patients; to the upper one-third in 53 patients; to the upper one-half in 55 patients; to the upper two-thirds in 75 patients; to an entire lung in 87 patients; to only the middle third in one patient; to the lower one-half in one patient; to the lower two-thirds in two patients; and one patient could not be classified.

One or more cavities were seen on the preoperative roentgenograms of 236 patients (fourteen of these patients had a complicating empyema). Among the 278 patients, cavities which were visible at one time were no longer definitely recognizable in twenty-six. Sixteen patients never had a visible cavity (planigrams were not in use at this clinic prior to 1946). A majority of patients (151, or 57.9 per cent) had one cavity. Table II demonstrates the number of cavities shortly before operation.

Table III lists the variations in the greatest diameter of the cavities. The patient with the largest cavity (12 cm.) was alive eleven years after thoracoplasty, had negative sputum cultures, and no cavity was visible on the roent-

TABLE II. NUMBER OF CAVITIES SEEN PREOPERATIVELY

NUMBER OF CAVITIES	NUMBER OF PATIENTS
None	42
1	151
2	24
3	14
4	6
5	1
More than 5	12
Moth-eaten appearance	18
Unknown	10

genogram. The majority of the cavities were round or oval and had moderate to extensive parenchymal infiltration about them. Ten of the cavities were classified as partly blocked and fluid levels could be demonstrated in all of them.

TABLE III. CAVITY SIZES

CAVITY SIZE	NUMBER OF PATIENTS
No cavity	42
Less than 1 cm.	24
Less than 2 cm.	51
Less than 3 cm.	53
Less than 4 cm.	50
Less than 5 cm.	28
Less than 6 cm.	17
6 to 8 cm.	9
8 to 10 cm.	3
12 cm.	1

In the contralateral lung, the tuberculosis was classified as minimal in 83 patients, moderately advanced in 103 patients, far advanced in 44 patients, and no disease in 48. As previously mentioned, the contralateral lesions were active in 115, and 15 patients had visible cavities. Seven additional patients had had cavities in the so-called ''better lung'' but they were not visible on the immediate preoperative films. The greatest number of cavities in this group was 2, and the largest was 7 cm. in diameter.

The data mentioned above are listed merely to serve as an indication of the various types of lesions found in this 278-patient group. A correlation between these data and the results is not presented since there would be too few patients for the many divisions.

Tuberculous bronchitis was noted preoperatively in 76 patients. A detailed study of this group is still being carried on even though a group this small may not be of statistical importance. Thirty of the patients had bronchial lesions on the side of the contemplated thoracoplasty which were very localized; 18 were moderately extensive; and 7 were very extensive. Ten patients had bilateral bronchial involvement with 7 of the lesions in the contralateral side described as very localized, 2 as moderately extensive, and one as very extensive. A decrease in the caliber of the involved bronchus was noted in 27 patients. The lumen of the bronchus was described as being reduced by 50 per cent or less in 17 patients, and 85 per cent to 95 per cent in 3 patients. Of the 76 patients with tuberculous endobronchitis, 53 were found at the time of this follow-up study to be alive and 23 were dead. Of those alive, 9 patients were working full time more than ten years after operation, considered themselves well, and all but one (with intermittently positive sputum cultures) had negative sputum; 20 were well and working full time more than five years after operation and had negative sputum; 10 were well but stated that they worked part time (7 had negative sputum, one became negative after a lobectomy, and 2 had intermittently positive sputum); 5 patients stated that they were short of breath (3 had negative sputum, 2 were positive on culture only, and one of the latter had a visible cavity beneath the thoracoplasty but refused further treatment); 8 patients failed to answer the questionnaire but all had had negative sputum cultures more than five years since thoracoplasty; one patient is still in the sanatorium with positive sputum and a draining cavernostomy. Thus, of the 53 patients still alive, 46 have negative sputum cultures and 35 consider themselves well and are working. Of the 23 patients who died, 5 lived more than five years after operation, 7 lived three to five years, 5 lived one to three years and 6 lived less than one year. The causes of death in this group are listed as follows:

12 patients had a spread or exacerbation of their disease and died of progressing tuberculosis;
3 patients died of cardiorespiratory decompensation;
2 died from accidental causes (aniline dye poisoning and pontocaine poisoning during anesthesia for bronchoscopy);
1 died as a suicide;
2 died from tuberculous meningitis;
1 died of a bronchoesophageal fistula which required gastrostomy;
1 died from the combination of an infected wound and atelectasis; and
1 died from an unknown cause.

To compare the results with the other patients who did not have tuberculous bronchitis would be misleading since these seventy-six patients are a highly selected group in which the types of tuberculous bronchitis that would contraindicate thoracoplasty are not represented.

The Semb extrafascial apicolysis was added to the thoracoplasty in 63 patients. Of these, 21 are dead and 42 are alive. An attempt was made to perform this procedure in 6 additional patients, but it was not possible to do so. Of these 6 patients, 5 are alive and one dead (not as a direct result of the attempt). Five patients in whom the Semb apicolysis was performed developed infection in the subscapular space and two of these are alive. We cannot be at all certain that the infections were a direct result of the procedure, since other factors which might have contributed toward the infection were present in all the patients.

In a majority of the 278 patients the indication for the thoracoplasty was cavitary disease (216 or 77.7 *per cent*). Other indications were: failure of pneumothorax (or any other treatment) to control the disease (no cavity being visible), 35 patients (12.6 per cent); empyema with a cavity in the lung, 16 patients or 5.7 per cent; empyema without parenchymal cavity, 7 patients or

2.5 per cent; to obliterate an extrapleural space (not empyema), 2 patients or 0.7 per cent; repeated hemoptysis (sputum negative), one patient; unexpanded lung under pneumothorax space, one patient.

The number of ribs removed varied according to the extent and type of the lesion. Table IV lists the number of ribs from which segments were removed.

TABLE IV. EXTENT OF THE THORACOPLASTY

NUMBER OF RIBS	NUMBER OF PATIENTS
3	13
4	20
5	25
6	29
7	67
8	72
9	31
10	16
More than 10	5

To obtain better collapse where fewer than seven ribs were removed, a portion of the scapula was removed in 31 patients. A complementary anterior thoracoplasty of the Haight type[3] was performed in 158 of the 278 patients. The majority of these included three or four ribs (144), but in 4 patients two ribs were removed, in 5 patients five ribs were removed, in 2 patients six ribs were removed, and in 3 patients seven ribs were removed. Formalin (10 per cent) was applied to the periosteum after the ribs were removed in order to delay or prevent regeneration of the periosteum. This was done in all cases only in the area between the axillary border of the scapula and the bases of the transverse processes. Care was exercised not to apply the formalin anterior to the axillary border of the scapula where it might result in a soft chest wall and complicate the postoperative convalescence. In practically every instance the interval between stages was three weeks. In a large majority of patients the anesthetic agent was nitrous oxide-oxygen-ether, supplemented by intercostal nerve block with 1 per cent procaine.

Accidents occurring during operation were few. The parietal pleura was torn in six patients; the internal mammary artery and vein and the first intercostal artery were each torn once; a root of the brachial plexus was injured once (recovery was complete); and an empyema space was entered once.

Postoperative convalescence was studied under two headings; parenchymal and extraparenchymal. In the former category, atelectasis occurred on the side of operation in 2 patients, spread or reactivation of the disease occurred in 34 patients. A detailed report on this subject has been made by Yang and Lees[4]; two-thirds of the patients survived the spread, and about one-third died. The extraparenchymal complications consisted of: pleural effusion on the side of the thoracoplasty in 6 patients; effusion on the contralateral side in 3 patients; empyema on the side of operation in 3 patients; tuberculous wound infection in 6 patients; pyogenic wound infection in 25 patients (many of these were localized stitch abscesses which healed readily); wound hemorrhage shortly after operation (controlled with pressure dressings) in 2 patients; a febrile postoperative course (no pus noted) in 6 patients. Scoliosis at the time of follow-up was minimal in 59 patients, mild in 66 patients, moderate in 104 patients, severe in 19 patients, and none was noted in 30 patients. None of the patients with severe scoliosis was disabled by it.

Seventy-five (27.0 per cent) of the 278 patients are dead. Twenty-three of these died more than five years after thoracoplasty. Table V lists the time of death after the last stage of the operation, and Table VI lists the causes of death.

TABLE V. TIME OF DEATH AFTER OPERATION

LENGTH OF TIME	NUMBER OF PATIENTS
Less than 3 months	10
3 to 6 months	8
6 to 12 months	5
1 to 2 years	10
2 to 3 years	8
3 to 5 years	11
5 to 13.5 years	23

TABLE VI. CAUSES OF DEATH

Progressive tuberculosis on unoperated side	17
Exacerbation of lesions on operated side	9
Spread to lung on operated side	6
Cardiorespiratory decompensation	14
Extrapulmonary tuberculosis	6
Nephritis and amyloid disease	4
Tuberculous meningitis	3
Suicide	3
Accidental death by poisoning	2
Operative complications	2
Extrapulmonary carcinoma	2
Leucemia	1
Unknown	6

Thus, from Table VI we note that 17 patients died of progressive tuberculosis in the lung on the unoperated side, 15 patients died of progressive tuberculosis of the operated side, and 14 of cardiorespiratory decompensation. Among the 6 patients who died of extrapulmonary tuberculosis, 4 had active pulmonary lesions and 2 appeared stable shortly before death. Tuberculous meningitis claimed 3 patients; one died six weeks after the last stage of his thoracoplasty; thoracoplasty was performed for a second patient to prevent continued seeding of lesions on the better side which were clearing satisfactorily, but a spread and reactivation occurred. Death from the meningitis came three months afterward; the third patient died four months following the last stage of his thoracoplasty. Death from poisoning occurred in one patient from absorption of aniline dye used industrially and in another patient during anesthetization with pontocaine solution prior to a check-up bronchoscopy for a previously existing tuberculous bronchitis. One of the two deaths from operative complications occurred in a patient who was having a thoracoplasty performed over a previously performed extrapleural pneumothorax which did not control the tuberculosis. A bronchopleural fistula developed and was listed as the cause of death; the other death was due to bilateral atelectasis in a patient who would not or could not cooperate in raising his sputum and died twenty-four hours after operation.

Thirty-three patients died twenty-four hours to two years after the thoracoplasty; 15 from progressive tuberculosis after an exacerbation or spread; 6 from cardiorespiratory decompensation; 3 from tuberculous meningitis; 2 from operative complications; 2 from suicide; 1 from nephritis and amyloidosis; and 4 from unknown causes.

Forty-two patients died at varying intervals from two years to thirteen and one-half years. The patient, who lived thirteen and one-half years after operation, died of carcinoma of the uterus. Eight patients died two to three years after operation (3 from progressive tuberculosis in the unoperated lung, one from an exacerbation in the operated side, 2 from cardiorespiratory decompensation, 1 as a suicide, and 1 from nephritis and amyloidosis); 11 patients died three to five years after operation [8 died from nontuberculous causes as follows: cardiorespiratory decompensation (1), accidental poisoning (2), acute appendicitis (1), neurosyphilis (1), pneumonia (1), leucemia (1), and hypertensive heart disease (1); and 3 died from progressive tuberculosis]; 23 patients died in from five to thirteen and one-half years (10 from progressive tuberculosis, 4 from cardiorespiratory decompensation, 1 from intestinal tuberculosis, 1 from a cerebral-vascular accident, 1 from a bronchoesophageal fistula, 1 from hypertensive heart disease, 1 from a nontuberculous pneumonia, 2 from extrapulmonary carcinoma, 1 from severe diabetes, and 1 from an unknown cause). Of this last mentioned group of 23 patients 11 were discharged from the sanatorium as apparently arrested, and later died of nontuberculous causes. Four were discharged against medical advice with quiescent disease, and later died of progressive tuberculosis; 4 were discharged as apparently arrested and died later of reactivated lesions; 1 was discharged as quiescent (intermittently positive sputum cultures) and died later of intestinal tuberculosis; and 3 patients were never discharged and died in the sanatorium of tuberculosis.

Nineteen of the 75 patients who are dead had negative sputum at the time of the follow-up examination. However, 11 of these 19 had pulmonary or intestinal lesions which appeared active by x-ray examination. We can say, therefore, that only 8 of the 75 patients probably had truly negative sputum. The causes of death listed for these 8 patients are: carcinoma of the uterus, 1; severe diabetes, 1; nontuberculous pneumonia, 1; cerebral-vascular accident, 1; and cardiac disease, 4.

If we consider three months after operation as the maximum period of time during which death may be attributed directly to the thoracoplasty, there were 10 such patients, an early mortality of 3.6 per cent. Among the 65 patients who died more than three months after operation, 26 (9.4 per cent of 278 patients) died of nontuberculous causes, 33 (11.9 per cent of 278 patients) died of tuberculous causes, and 6 died of unknown causes.

Of the 203 living patients, 6 had lesions which never healed. Three of these were bronchoscoped and 2 were demonstrated to have tuberculous bronchitis. The third patient was believed to have an invisible cavity, and eight years after thoracoplasty had a lobectomy with prompt conversion of the sputum and persistently negative cultures, during the one and one-half years since the lobectomy. This patient is, of course, considered as a thoracoplasty failure. Three patients were not bronchoscoped; two of them had had blocked cavities preoperatively, and tuberculous bronchitis was suspected as the cause of the unhealed lesions. The third patient was known to have tuberculous bronchiectasis.

Interpretation of the roentgenograms of the 203 living patients from five to fifteen years after the last thoracoplasty stage showed that 181 (65.1 per cent) of the whole group of 278 patients and 89.1 per cent of the 203 living patients apparently had inactive lesions, and without cavitation. Twenty-two patients apparently had roentgenologically active lesions.

One hundred seventy-four patients had negative sputum cultures at the time of the follow-up examinations (62.5 per cent of the whole group of 278 and 85.7 per cent of the 203 living patients). These figures include 23 patients who had no sputum (although when it had been present it was negative on three successive monthly cultures) and who did not have follow-up cultures of gastric contents either because they refused or because it seemed impractical to require them to go without breakfast, or without breakfast and lunch before making a more or less long trip to the sanatorium Outpatient Department. In addition to the 174 negative patients, there were 8 patients who died from causes unrelated to tuberculosis (as listed above) and whose follow-up cultures had been negative; if these 8 are added to the 174, as we believe they should be, 182 (65.4 per cent of the whole group of 278 patients and 89.6 per cent of the 203 living patients) have or had negative cultures.

Upon reviewing the patients' answers to the questionnaires certain information was gathered. Nineteen patients, however, failed to answer or sent inadequate answers; we learned indirectly that 5 of them were apparently well and were working; the data about these 19 patients were therefore obtained from the records of their last examinations, all of which were five years or more after operation.

Among the 203 living patients, 134 (66.0 per cent) considered themselves well, and they were doing full-time work. Forty-four additional patients also considered themselves well but 23 (11.3 per cent) were doing only part-time work and 11 (5.4 per cent) stated they could not work (the majority of these were more than 60 years of age). Ten patients were still in the sanatorium when the study was completed; several of them have since had resection operations, the results of which are not included in this report. Six patients are living but not well; 3 of these six patients have negative sputum and apparently healed lesions by roentgenogram, but they are dyspneic.

Since the study was completed in 1949, we did not utilize the 1950 NTA classification. Utilizing the National Tuberculosis Association's classification in force prior to 1950, the 278 patients may be listed as follows:

Apparently cured	140 (50.3 per cent)
Arrested	9 (4.3 per cent)
Apparently arrested	36 (12.9 per cent)

Quiescent	2 (0.7 per cent)
Improved	11 (3.9 per cent)
Worse	5 (2.0 per cent)
Dead	75 (27.0 per cent)

SUMMARY

1. Two hundred seventy-eight patients who had the modern type of thoracoplasty were followed from five to fifteen years after operation.

2. Little difference was noted with regard to sex or side of operation, there being about 50 per cent of each.

3. No streptomycin or penicillin was used in the group either before or after operation.

4. The major indication for thoracoplasty was cavitary tuberculosis.

5. Seventy-six patients had tuberculous bronchitis; 53 of these patients are alive (46 have negative sputum); 23 patients are dead (5 lived more than 5 years after operation).

6. Spread or reactivation of disease occurred in 34 patients. Of these about two-thirds survived and one-third died.

7. Seventy-five (27.0 per cent) of the 278 patients are dead.

8. Two hundred three patients are living; 174 (85.7 per cent) of them have negative sputa. If the 8 patients who died from nontuberculous causes but who had negative sputa are added, the figure is 89.6 per cent of the 211 patients.

9. According to the NTA classification in use when this study was completed (1949), the 278 patients may be listed as follows: apparently cured, 140 or 50.3%; arrested, 9 or 4.3%; apparently arrested, 36 or 12.9%; quiescent, 2 or 0.7%; improved, 11 or 3.9%; worse, 5 or 2.0%; dead, 75, or 27.0%.

We wish to express our appreciation to Dr. Edward W. Laboe, formerly director of the Michigan State Sanatorium, for his splendid cooperation in placing at our disposal the laboratory and clerical facilities required. In addition, acknowledgement is made of the services of the late Dr. Edward Finch who examined those patients who returned to the Outpatient Department of that sanatorium and who aided in the interpretation of the films mailed to the sanatorium.

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DISCUSSION

COL. JAMES H. FORSEE, Denver, Colo.—This splendid paper so well presented by Dr. Lees is a distinct addition to our information relative to follow-up data pertaining to thoracoplasty. We have recently undertaken an attempted survey of thoracoplasties performed over a long period, from 1927 through 1946. This, of course, represents various types of thoracoplasties performed during those years, and we have had the very valuable assistance of the National Research Council in making this follow-up study.

(Slide.) There is a total of 514 patients, military personnel, who have been followed at a 97 per cent follow-up rate. There is one thing very positive in the interpretation of such data and that is whether or not the patient is dead or alive. Forty-seven per cent of the entire group are dead.

(Slide.) Following the era of streptomycin, beginning in 1947, we have the following data, of course in a much shorter follow-up period, and doubtless as we continue to follow these patients we will find that more and more deaths will be added to that column. However, of 172 patients operated on in 1947 and 1948, 9 per cent are dead. If we eliminate those operated on for tuberculous empyema the death rate is 6 per cent.

We have employed the type of thoracoplasty standardized by Dr. Alexander and his group, and we have not found it desirable to date to combine thoracoplasty at the time of lobectomy.

DR. ROBERT PANNIER, Bruges, Belgium.—I am very grateful to the Association for allowing me to take part in this discussion.

In relation to Dr. Lees' very interesting paper, I should like to say a few words on the late results of thoracoplasty as they came out of a recent follow-up of 1,137 cases operated upon between 1926 and 1948, in the chest department run by Dr. DeWinter at the St. John's Hospital in Bruges (see *Acta Tuberc. Belgico,* 1950). In so far as indications and operative technique are concerned, this is a very uniform group because all the cases have been selected by one man, Dr. DeWinter, and were operated upon by only two surgeons, the late Professor Sebrechts, until 1948, and then by one of his former assistants, Dr. Deems.

The technique has changed very little since 1930. Time does not permit me to go into details. However, I should like to stress two points: first, statistics compiled year by year show that in the group of patients who have successfully undergone thoracoplasty (this means that they were still alive three months after operation) most of the later deaths occurred in the first three years following the operative procedure. These figures are naturally more severe in the poor risks than in the good risks. Those who have successfully undergone that period of consolidation, as we call it now, may expect a long survival.

7 patients are alive for more than 23 years
9 patients are alive for more than 21 years, and
7 patients are alive for more than 20 years.

These were not all good risks and it is interesting to note that of that small group of 22 patients, 4 were poor risks, 5 severe cases with extensive bilateral disease, and 3 had a bronchopleural fistula with empyema.

In 1949, the results for the entire group of 1137 were as follows:

Of the good risks	70 per cent are alive for 23 to 3 years after operation
Of the moderate risks	57 per cent
Of the poor risks	27 per cent
Of extensive bilateral disease	33 per cent
Of bronchopleural fistula and empyema	26 per cent

The second point I should like to stress is that those who die after that three-year consolidation often die of tuberculosis, but not necessarily of pulmonary tuberculosis. Some have been well and working for ten to twelve years after operation.

I am very sorry not to be able to show the photograph of a group of 31 patients who were presented at a meeting in 1931, who had had their thoracoplasty one to four years previously. I have no slide with me, as I did not expect to have an opportunity to present it here, and there is unfortunately no epidiascope available. In 1950, 22 of these 31 patients are still alive and well, now for more than nineteen years. Of the 9 who died, one died in an accident in 1935; one died of heart failure in 1945; and seven died of tuberculosis

1 after	7 years
1 after	9 years

2 after	10 years
1 after	11 years
1 after	12 years
1 after	17 years

This shows that we do not always cure the patient—in many cases we only arrest the disease; sometimes, however, for many, many years.

DR. CAMERON HAIGHT, *Ann Arbor, Mich.*—As you know, Dr. Alexander's group has been very much interested in the subject of thoracoplasty. We believed when this study was undertaken that it would be timely to learn the late results of thoracoplasty, particularly in view of the increasing use of pulmonary resection, so that it would enable a comparison of the late results of thoracoplasty with at least the early results of resection. There are two points that I would like to stress in connection with the cases Dr. Lees has presented.

It is well known that the results of thoracoplasty can be considerably influenced by the selection of cases that give the most promise of success. In this regard I would like to say that the cases which Dr. Lees has shown have been chosen not because it was felt that they would necessarily obtain a good result, but in a number of instances because nothing else could be done. We have often accepted what we consider to be extremely poor risks for thoracoplasty, including patients with a very low respiratory reserve in whom it was problematic if the desired amount of collapse could be obtained. It is interesting, then, to see the late results in cases which include many substandard indications.

In the evaluation of any type of therapy for tuberculosis, the longer the cases are studied the less satisfactory the results will be. This was the reason that patients operated on in the last five years have not been included, as it was desired to study only late results. This series illustrates the decreasingly favorable results with the passage of time and, therefore, the results cannot be compared with those obtained within three to five years after operation.

At the present time we continue to rely greatly on the use of thoracoplasty in the treatment of pulmonary tuberculosis. The actual number of cases of thoracoplasty, however, is less than formerly, inasmuch as approximately one-half of the patients for whom major surgery is indicated are now treated by resection. A problem which bothers us greatly at present—and I believe that the answer has not yet been reached—is the choice of procedure in the treatment of large inflation cavities; that is, whether resection should be done or whether they should be treated by thoracoplasty with the hope that the thoracoplasty will be effective. At present, we are using the latter plan with certain exceptions, thereby reserving resection for those cases in which thoracoplasty has failed.

DR. LEES (closing).—I wish to thank the discussors. Unfortunately, time did not permit going into great detail about the causes of death; they will be listed in the paper.

With regard to Dr. Pannier's mention that the late cause of death in the majority of instances in his study was due to tuberculosis, our results are not entirely thus. Approximately one-third of the patients died within a year, those deaths being due, in the main, to progressive tuberculosis. Approximately one-third of the patients died within one to three years, in the majority of instances from tuberculous causes. The rest of the patients, 34, died three years or more after thoracoplasty. Of these last mentioned patients, 10 died of tuberculosis; eight from progressive tuberculosis in the contralateral lung, and two from reactivated tuberculosis beneath the thoracoplasty.

SIMULTANEOUS DECORTICATION AND RESECTION IN UNSUCCESSFUL PNEUMOTHORAX

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THE problem of unexpandable lung after pneumothorax and its complication, tuberculous empyema, is a serious one. Review of the literature reveals that 50 to 70 per cent of all pneumothoraces develop fluid at some time.[15, 23] Furthermore, following pneumonolysis, about 50 per cent of the patients develop more or less persistent fluid.[15] The irritating effect of this fluid results in the development of a fibrous membrane on the visceral and parietal pleuras which sometimes prevents re-expansion of the lung, when pneumothorax treatment is discontinued. The incidence of unexpandable pneumothorax has been quoted as 5 to 10 per cent.

Accepting the philosophy of Robin[22] that all pleural effusions in tuberculous patients have a tuberculous etiology and that adequate search will reveal organisms, it is not surprising that 15 to 20 per cent of such effusions develop finally into empyema.[15, 23] Similarly, following pneumonolysis, about 6 per cent of cases go on to tuberculous empyema.[15] This tuberculous empyema membrane likewise prevents re-expansion of the lung and results in marked impairment of function of diaphragm and chest wall. A further serious complication of empyema is the development of bronchopleural fistula which, due to aspiration of empyema fluid, often leads to contralateral spread and ultimate fatality.

Following discontinuance of pneumothorax, the lung frequently does not re-expand appreciably in these complicated cases. The pleural space, and rarely the empyema space, may be obliterated, but this is accomplished more by the shift of the mediastinum, elevation of the diaphragm, and flattening of the chest wall than by lung re-expansion per se. The disadvantages of such a mechanical situation are well known. They are overdistention of the contralateral lung with associated danger of reactivation; dysphagia due to retraction of the esophagus; deformities of the thorax; and last but not least, limitation of respiratory function. Therefore the mere obliteration of pneumothorax space is not always the desired end.

Previous attempts at solving these problems resulting from pneumothorax complications have not always been satisfactory. Thoracoplasty has been successful if the membrane preventing re-expansion has been thin. Here, however, considerable healthy lung is collapsed and thus lost from function in achieving obliteration of the space. More often, however, a total thoracoplasty is not successful in completely obliterating the space when the membranes are

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thick, as is usually the case in tuberculous empyema. Even if they are in contact, the opposing surfaces will frequently not fuse because of avascularity of the membranes and the presence of tuberculous granulations and caseous areas in them. When active parenchymal disease persists in the unexpanded lung and is covered by a dense membrane, it can seldom be collapsed further by thoracoplasty. Thus thoracoplasty has little or no effect on active disease under these circumstances, as well as little or no effect on obliterating the residual space.

Schede thoracoplasty has also been used to attempt to obliterate tuberculous empyema when thoracoplasty alone was unsuccessful or when bronchopleural fistula was present. This operation is a mutilating procedure with considerable resultant deformity of the chest wall. It is a bloody and shocking operation with a high operative mortality, requiring multiple procedures, and carrying a considerable morbidity. The operation, therefore, is conscientiously avoided by most thoracic surgeons.

Decortication as the modern concept of the treatment of unexpandable lung with its complications has evolved from the contributions of various surgeons in the past. Great impetus was given by the experience of surgeons in World War II with chronic clotted and infected hemothorax. The principles of decortication were then applied to other forms of chronic unexpandable lung such as chronic nontuberculous spontaneous pneumothorax and pyogenic empyema.[1] These rapid strides in development were aided by improvements in technique, anesthesia, blood replacement, and antibiotics. With the establishment of the value of decortication in nontuberculous conditions on a firm basis, it was but a short step to apply the same principles to the unexpandable tuberculous lung.

PATHOLOGY

The pathogenesis of the unexpandable lung is well recognized, and has been clearly stated by Weinberg and Davis.[7] The pathologic changes may vary greatly, and for our purposes five points will be discussed briefly. First is the earliest stage of the unexpandable lung with opacity of the pleura without other gross change. Microscopically it is a thin fibrinous membrane richly vascularized and infiltrated with leucocytes and histiocytes. This is probably a reversible phase. There is the later second stage when a gross membrane becomes visible which may vary from 2 to 3 mm. in thickness in the visceral layer and from 3 to 10 mm. in thickness in the parietal layer. On microscopic examination there is a hyalinized fibrous middle layer with avascular peripheral layers. Although typical tuberculous lesions may be found in all layers, they are not always present. The fluid in the space may be clear or turbid and may or may not contain tubercle bacilli. A pleural endothelial layer may be recognized. This stage is usually irreversible. In the third stage frank empyema may be present with a thick membrane lined by fibrinous and caseous exudate. Microscopically there is an absence of an endothelial layer, but there is a hyalinized middle layer of the membrane showing all stages of inflammation and degeneration from endothelial tubercles to caseation and calcification. This membrane may be densely adherent by fibrosis to pleura and lung. The sac usually contains purulent fluid from which tubercle bacilli can readily be isolated. Fourth, the membrane, because of its constricting nature, may produce infolding of the lung. This, plus collapse, does not produce parenchymal fibrosis, but fibrosis when present is related to the inflammatory process in the parenchyma and not to the collapse per se. Fifth, the parenchymal pathology will depend upon the state of activity of the tuberculosis. This pathologic picture is well known and does not warrant repetition here.

INDICATIONS AND CONCEPTS OF TREATMENT

The indications for decortication in pulmonary tuberculosis have been discussed frequently in the past few years[3-10] and involved primarily arrested or healed parenchymal disease associated with an unexpandable lung. We accept these as indications for decortication, but believe that there are other fundamental principles which must also be considered. Of primary importance in any case is the status of the underlying lung. Here must be considered the bacillary content of the sputum, activity of the tuberculous focus, and the extent of parenchymal involvement. A positive sputum established as arising from the encased lung is a warning that simple decortication will not be definitive, and further may be disastrous by way of spread. The uncontrolled parenchymal focus assumes major significance here and its treatment must be a part of the total therapeutic plan.

Extent of parenchymal involvement prior to induction of therapeutic pneumothorax should always be defined prior to decortication. Large necrotic and cavitary foci, seemingly arrested by pneumothorax, may reactivate upon decortication. Likewise extensive caseous foci may produce localized peripheral fibrosis between peel and lung, thus technically preventing decortication. This consideration must be a part of the total therapeutic plan.

Further, widespread parenchymal involvement may produce sufficient fibrosis to prevent maximum re-expansion postoperatively, resulting in failure to fill the pleural space. This situation, as is well known, predisposes to increase postoperative complications and decortication failures. Thus the relationship of pleural space to available lung volume must be a part of the total therapeutic plan.

The importance of these fundamental principles in the preoperative analysis of the case has resulted in a reclassification of indications for decortication in tuberculosis, as shown in Table I, and concepts in treatment in Table II.

TABLE I. INDICATIONS FOR DECORTICATION IN TUBERCULOSIS

I. Successful pneumothorax—sputum negative
    A. Peel, with or without fluid
    B. Tuberculous empyema
II. Unsuccessful pneumothorax—sputum positive
    A. Peel, with or without fluid
    B. Tuberculous empyema with or without bronchopleural fistula
III. Tuberculous pleurisy with effusion (spontaneous)
    A. Unexpanded lung
    B. Empyema

TABLE II. CONCEPTS IN TREATMENT

- I. Successful pneumothorax
  - Complete re-expansion undesirable or impossible because
    - A. Extensive parenchymal disease—danger of reactivation
    - B. Perpendicular fibrosis (lung—peel)
  - Treatment: 1. Preliminary tailoring thoracoplasty
    - 2. Decortication—uninvolved lung only
- II. Unsuccessful pneumothorax
  - Uncontrolled parenchymal disease
  - Treatment: 1. Preliminary tailoring thoracoplasty—except in segmental or block
    - 2. Resection—block, segmental, lobe or lung
    - 3. Decortication uninvolved lung

In respect to indications, the first group includes those cases usually accepted *as an indication for decortication, namely, unexpandable lung* with parenchymal disease arrested and sputum negative. In this group we have, however, made an important subdivision distinguishing those arrested or healed cases with such extensive parenchymal involvement prior to the therapeutic pneumothorax that re-expansion of the involved areas is not desirable because of danger of reactivation. It is believed that here permanent collapse is necessary to insure stabilization of the diseased area. One author has indicated that in a statistically significant series of pneumothorax follow-ups it was determined that where the parenchymal cavity is more than 4 cm. in diameter, the expected cure rate by pneumothorax treatment will not be over 4 per cent. It is this group of patients in whom we believe that permanent collapse should be instituted to insure the future health. Also the extensive disease may have resulted in such widespread fibrosis of peel to parenchyma that decortication is technically difficult if not impossible, and in such a case a tailoring thoracoplasty is necessary to obliterate residual pleural space. In this situation preliminary tailoring thoracoplasty is done and usually consists of subperiosteal resection of the first two and shorter portions of the third and fourth ribs. As soon as the chest wall is stabilized (in three to four weeks) decortication of the healthy portion of the lung is carried out leaving the diseased areas untouched.

The second basic division of cases of unexpandable lung includes those with positive sputum due to a varying extent of active uncontrolled pulmonary disease despite the adequate anatomical collapse of a pneumothorax. Permanent collapse by thoracoplasty *is not* indicated, *since apparently adequate collapse* by pneumothorax was ineffective in controlling the disease. Further, thoracoplasty sufficient to collapse the diseased area maximally would needlessly collapse healthy lung tissue. In these cases, preliminary tailoring thoracoplasty is at first performed if more than a block or segmental resection is contemplated. After the same short interval resection of the involved parenchyma is done and the healthy portions of the lung are decorticated. If pneumonectomy is necessary, due to bronchial disease or destroyed lung, it is done without prior thoracoplasty together with resection of empyema sac if present. Thoracoplasty follows after a short interval (three to four weeks) to diminish the size of the pleural cavity. In both of these basic groups the presence of tuberculous empyema rather than sterile fluid and fibrous peel does not alter the treatment.

The third and minor group is the occasional instance when the ordinary spontaneous tuberculous pleurisy with effusion is not absorbed as usual but goes on to the development of unexpanded lung or empyema. In these cases parenchymal foci usually are not demonstrable and sputum is negative. The treatment here is simple decortication of the lung and chest wall, and occasionally a small parenchymal focus may be found subpleurally which may be resected.

OBJECTION TO DECORTICATION

There have been several objections voiced to decortication. First has been the fear of reactivation of the parenchymal tuberculous focus after re-expansion. Since pneumothorax was originally intended as a reversible procedure, and if it had been applied on sound indications, then this type of surgical re-expansion should be no more dangerous than the usual medical methods. Areas of extensive prepneumothorax disease should not be decorticated but should be kept collapsed permanently. It is undesirable to re-expand these areas and then treat them after reactivation occurs, but rather thoracoplasty as a tailoring procedure should be utilized followed by decortication of the healthy areas. Likewise in the case of persistent active disease the excision of this collapsed, but uncontrolled, disease is indicated combined with decortication. Tailoring thoracoplasty may also be indicated here. Again the importance of considering the extent and activity of the parenchymal disease prior to decortication is emphasized.

Second, the fear of chest wall infection resulting from entering tuberculous tissue perhaps has been a deterent to the operation. Experience, however, has shown that it rarely occurs. Likewise residual empyema is an infrequent complication. These complications have been minimized by the use of streptomycin and by obtaining complete re-expansion postoperatively as soon as possible. In this latter connection the use of preliminary tailoring thoracoplasty in selected cases is advantageous in addition to its value in regard to maintaining permanent collapse of extensive parenchymal lesions. Even with pneumonectomy and empyema resection, which occasionally of necessity is incomplete, residual empyema is uncommon. Parenthetically it should be said that all such procedures are followed by thoracoplasty to attempt to decrease pleural cavity volume.

TECHNIQUE

After careful study of a case, especially review of prepneumothorax roentgenographs, has satisfied our criteria for decortication, preoperative preparations include several details. Bronchoscopy is indicated to rule out bronchostenosis as an etiology for the failure in re-expansion. Streptomycin and PAS, if not already being administered, are given for at least one week and penicillin for four days preoperatively. Evaluation of the general condition is, of course, essential.

Operation is performed under general anesthesia using a posterolateral approach. The sixth rib is usually resected subperiosteally and a short posterior segment of an adjacent rib is also resected if the chest wall is rigid. In all cases it has been possible to excise parietal as well as visceral membrane. A cleavage plane can usually be developed between parietal peel and chest wall. This plane is relatively avascular and the peel strips easily laterally and anteriorly, but with greater difficulty at the point of reflection from visceral to parietal membrane. This is especially true at the posterior apex and the costophrenic angle. Excision of the parietal peel results in a more complete removal of tuberculous tissue in the case of empyema and in the return of more normal motion of the chest wall and diaphragm postoperatively.

Next, decortication of normal lung is done in the usual manner while the lung is partially inflated. Areas of diseased parenchyma previously planned for excision or permanent collapse are not decorticated. Excision is then carried out by individual ligation technique. Decortication and segmental type resection both produce raw surfaces with leakage of air, but since the resultant minute leaks are, for the most part, transient, no attempt at repair is made. When visible bronchiolar openings are present they are repaired. In those cases where preliminary tailoring thoracoplasty has been performed, the decorticated normal lung readily fills the pleural space without overdistention.

Adequate drainage for both air and fluid is of utmost importance in achieving early and complete re-expansion and associated obliteration of pleural space. One large (one-half inch wide diameter) fenestrated tube is placed through a stab wound inferiorly in the posterior axillary line. A second tube (a fenestrated No. 22 catheter) is placed to the apex of the pleural cavity through an anterior stab wound. Both tubes are connected to individual water-seal bottles. In selected cases active suction of 15 to 20 cm. of water is used, but generally simple water-sealed drainage has been satisfactory.

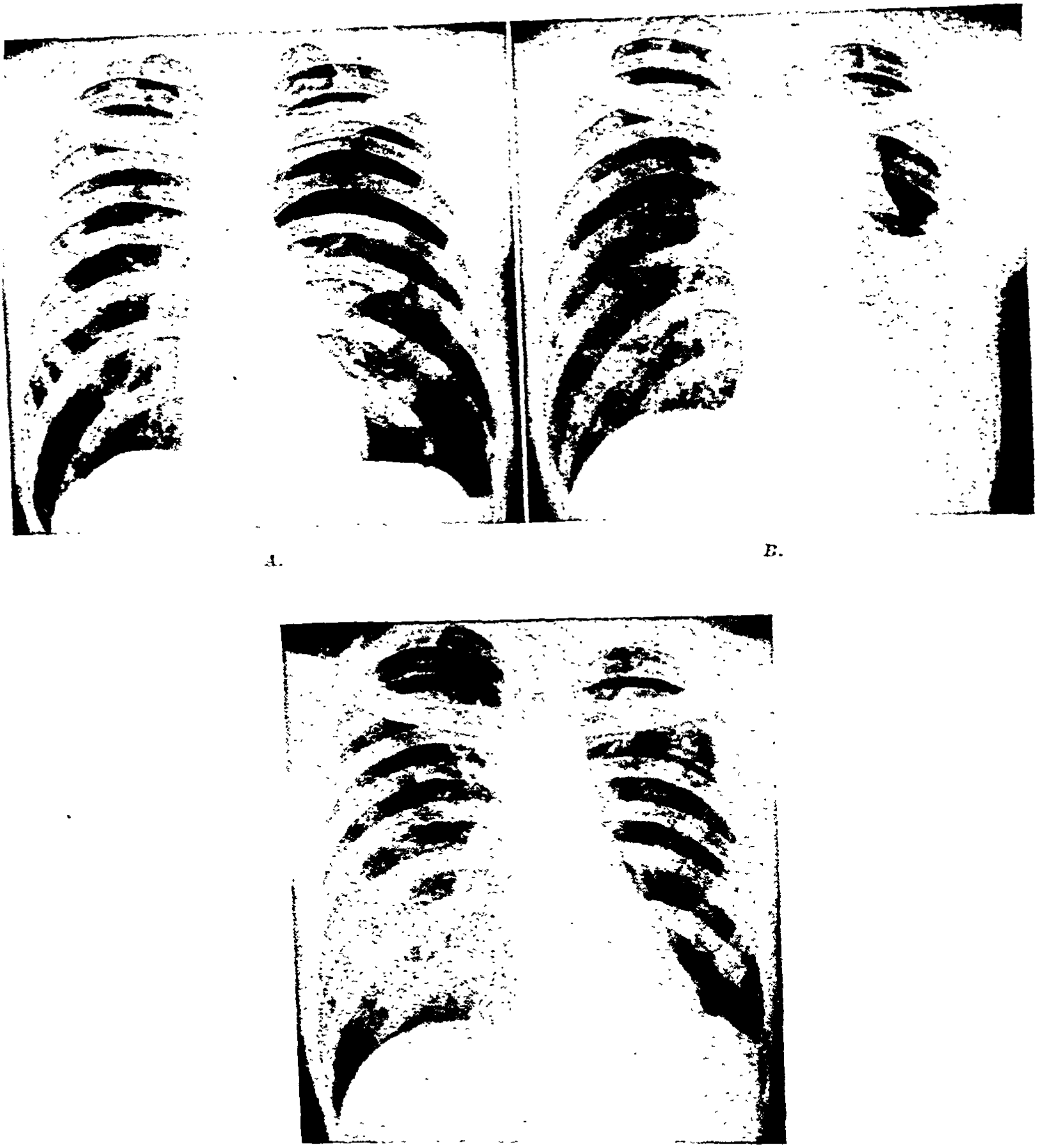
Postoperative streptomycin and PAS treatment is continued for a variable length of time and penicillin for seven to ten days. Of utmost importance is the prompt removal of tracheobronchial secretions. This is accomplished most satisfactorily by cough, but if cough proves ineffective, catheter tracheal suction is used as frequently as indicated. Bronchoscopy is seldom necessary, but may be resorted to. This removal of tracheobronchial secretions prevents development of localized atelectasis and, of course, insures prompt re-expansion and obliteration of pleural space.

RESULTS AND CASE REPORTS

Results in this series of fourteen cases are set forth in Tables IIIA and IIIB. A case illustrative of each indication in groups one and two is summarized briefly below:

CASE REPORTS

CASE 1 (Fig. 1 *A*, *B* and *C*.)—W. L., a 30-year-old white man, whose tuberculosis was diagnosed while in the Army in 1944. Left therapeutic pneumothorax was started in May, 1945, with pneumonolysis in July, 1945. He was ultimately discharged from the Army. In July, 1948, it was decided to abandon the pneumothorax. The left lung failed to re-expand, and increasing amounts of fluid developed. Aspiration of this fluid was done many times, and all laboratory findings were negative for tubercle bacilli. Decortication was done on Mar. 10, 1949, and postoperative convalescence was uneventful. He was discharged in July, 1949, gradually increased his activities, and at the present time he is working full time as an insurance agent. Sputum has remained negative by all methods including culture.



A.

B.

C.

Fig. 1.—*A*, Left pneumothorax. *B*, Unexpandable left lung; sputum, negative. *C*, After left decortication and re-expansion; sputum, negative.

This represents a case of moderately advanced original disease successfully treated with pneumothorax but complicated by unexpandable lung. Treatment here is simple decortication and re-expansion.

CASE 2 (Fig. 2 *A*, *B*, *C* and *D*.).—P. B., a 22-year-old white woman. Tuberculosis was diagnosed in 1943 and, after a short period of bed rest, right pneumothorax was begun in July, 1943. Fluid developed soon after the induction of the therapeutic pneumothorax. It was clear at first but soon became purulent, and was positive repeatedly for tubercle

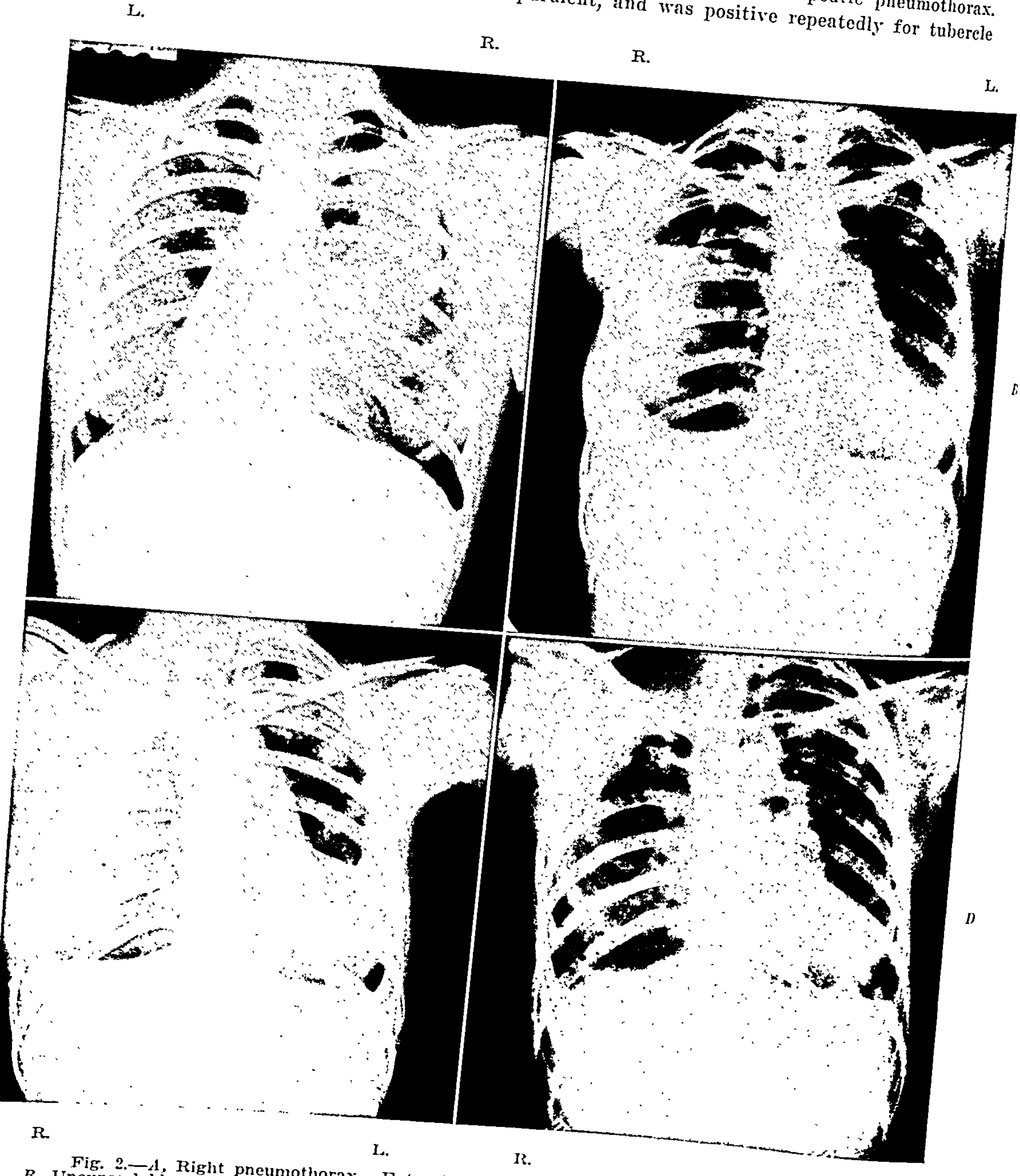
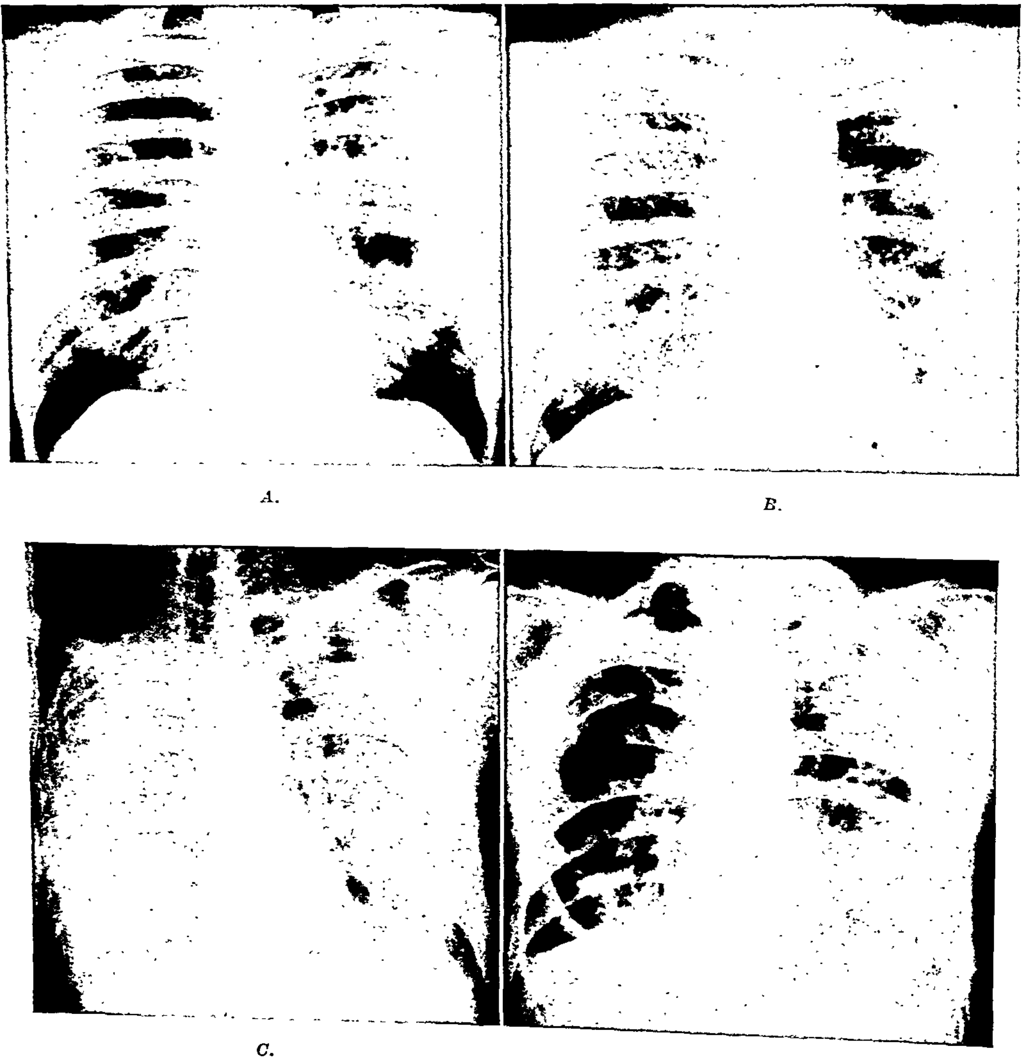


Fig. 2.—*A*, Right pneumothorax. Extensive right upper lobe disease with patent cavity. *B*, Unexpandable right lung with tuberculous empyema. Cavity closed and sputum negative. *C*, After preliminary tailoring right thoracoplasty. *D*, After decortication of right middle and right lower lobe. Right upper lobe premanently collapsed; sputum, negative.

bacilli. A left pneumothorax was begun in 1945 and was abandoned in August, 1949. Sputum had been negative on concentrate and culture since 1947. A preliminary tailoring thoracoplasty was done on June 16, 1950, consisting of the subperiosteal resection of the first and second, and shorter portions of the third and fourth, ribs. The transverse processes were not removed. Decortication of the right middle and lower lobes was done on July 14, 1950. The right upper lobe was not decorticated, and the liberated middle and lower lobes nicely filled the pleural cavity. The postoperative course was uncomplicated with sustained expansion of the middle and lower lobes, and the sputum has remained negative by all methods to date.



A. B.

C. D.

Fig. 3A-D.—A, Extensive left lung disease; left lung unexpandable. B, Right upper lobe disease appears. Left lung remains partially collapsed. C, bilateral unexpandable lungs with bilateral tuberculous empyema; sputum, negative. D, After right decortication; function much improved; sputum, negative.

This patient had extensive right upper lobe disease successfully treated by pneumothorax, but complicated by tuberculous empyema and unexpandable lung. Because of the original extensive disease, re-expansion of which was considered dangerous, thoracoplasty to maintain right upper lobe collapse, and decortication of the right middle and lower lobes were combined.

CASE 3 (Fig. 3 *A*, *B*, *C*, *D* and *E*.).—H. B., a 43-year-old man, first developed tuberculosis in 1932. In 1933 a left pneumothorax was induced for cavitary disease in the left upper lobe, with considerable surrounding infiltration. Pneumothorax was maintained until 1936. Following attempted re-expansion there was a considerable amount of fluid and pleural thickening, and the lung remained about 30 per cent collapsed. Right upper lobe tuberculosis was first present in 1945. In July, 1945, a right pneumothorax was begun and maintained until July, 1947, at which time a thickened pleura developed along with fluid. This lung remained about 40 per cent collapsed. Aspiration was carried out repeatedly on both sides and ultimately there developed a bilateral tuberculous empyema. Sputum, however, converted by concentration in August, 1947, and by culture in April, 1948. During the years dyspnea developed and the patient became a respiratory cripple. Anxiety attacks periodically would result in the need for oxygen administration. Bilateral decortication was discussed with this patient and he was anxious to take the involved risks. The right side had had the least amount of tuberculosis and it was therefore decided to start on this side, to restore as much pulmonary function as early as possible. Therefore, on May 31, 1949, right parietal and visceral decortication was carried out. Re-expansion was not prompt, but ultimately the pleural space was obliterated and this was associated with marked clinical improvement. The patient was now able to stroll in the corridor and anxiety completely disappeared. On Feb. 21, 1950, left parietal and visceral decortication was carried out. Upon the completion of this, it was noted that with maximum re-expansion the left lung would not completely fill the pleural space. Therefore an apical thoracoplasty was done at the same time, and again the patient recovered uneventfully. After a few months of modified bed rest, activities were increased and he is now ambulatory and is anticipating his discharge from the hospital. Sputum has remained negative except for a rare positive culture.

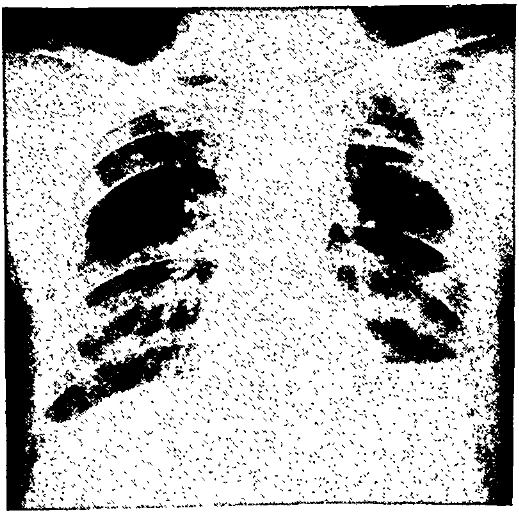
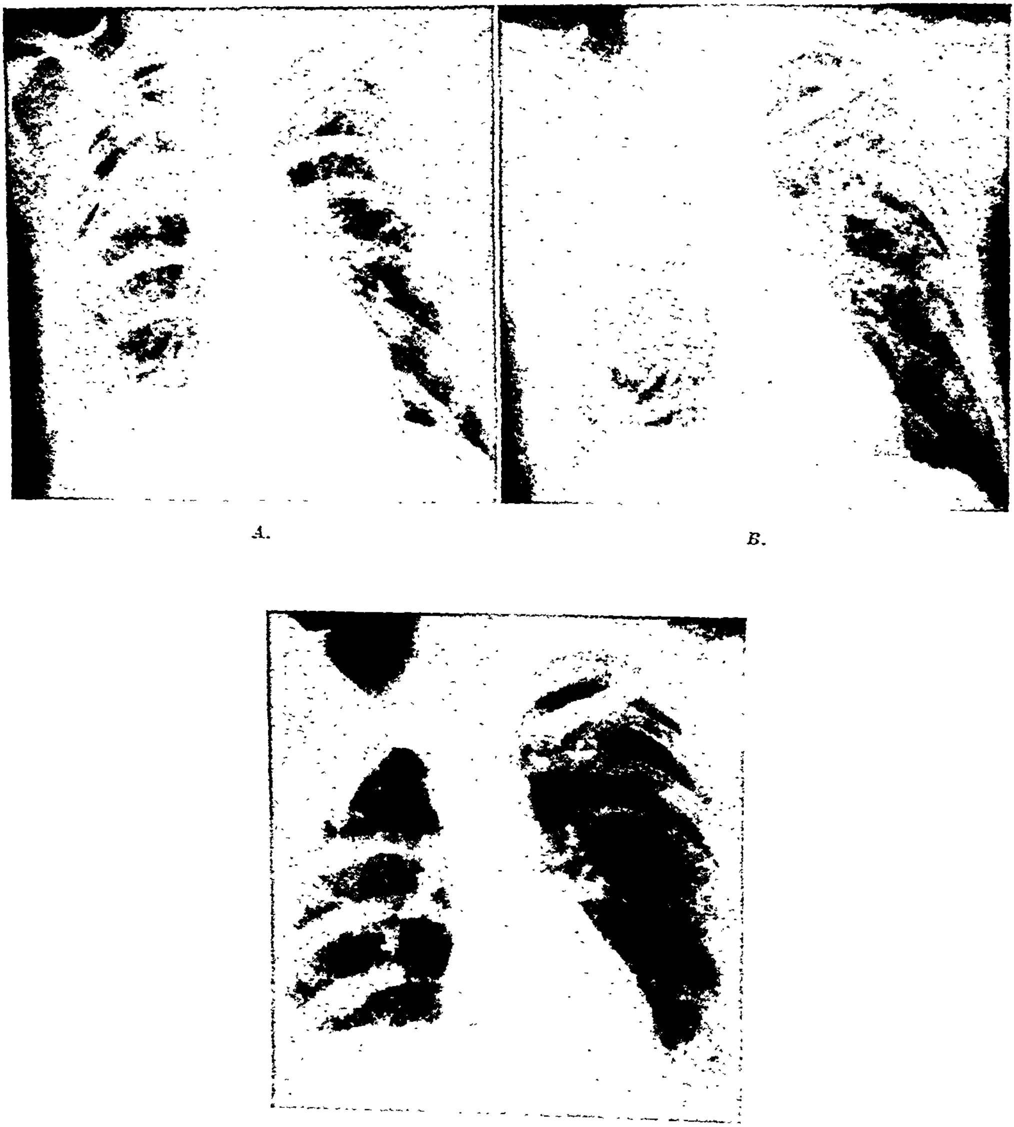


Fig. 3*E*.—After simultaneous left decortication and left thoracoplasty; function much improved; sputum, negative.

This is another case of extensive original disease successfully controlled by pneumothorax for which thoracoplasty combined with decortication was necessary on the left to prevent reactivation and to insure obliteration of pleural space. It is also of unusual interest in that it represents bilateral pneumothorax complicated by bilateral empyema resulting in a respiratory



A.

B.

C.

Fig. 4.—*A*, Unexpandable right lung with tuberculous empyema. Cavity patent in the right upper lobe, and sputum positive. *B*, After preliminary tailoring thoracoplasty. *C*, After right upper lobectomy and right middle and right lower lobe decortication; sputum, negative.

cripple. The combination of left thoracoplasty and bilateral decortication has changed a previously hopeless situation to one where the patient is now contemplating gainful employment.

CASE 4 (Fig. 4 *A*, *B* and *C*.).—G. H., a 35-year-old man, a hoisting engineer, developed cough and sputum in 1948. Chest roentgenograph in July, 1948, disclosed cavitary tuberculosis on the right, and positive sputum was found. He was therefore hospitalized, and a right pneumothorax was begun in August, 1948, but was complicated by pleuropulmonary adhesions. In December, 1948, a complete closed pneumonolysis was performed. This was complicated by fluid development which persisted and was positive for tubercle bacilli. The upper lobe became almost 100 per cent collapsed. Because of these complicating factors, re-expansion was attempted, but was attended by reopening of the cavity in August, 1949. Finally in December, 1949, an upper stage of thoracoplasty on the right was done. On Jan. 24, 1950, the right middle and lower lobes were decorticated, and the right upper lobe was excised. Except for one isolated culture, this patient's sputum has been negative by all methods since February, 1950. The spheroidal lesions in the contralateral lung have remained unchanged and stable. Block resection of these lesions was under consideration, but, because of disciplinary reasons, this patient was discharged from the hospital in July, 1950, and since that date has been working full time, is asymptomatic, and continues to be sputum negative by all methods.

Here, despite adequate anatomical collapse by pneumothorax, parenchymal disease was uncontrolled, with patent cavity in the right upper lobe and smear positive sputum. This was further complicated by unexpandable lung and tuberculous empyema. Sputum conversion followed right upper lobectomy and decortication of the right middle and right lower lobes preceded by a tailoring thoracoplasty.

CASE 5 (Fig. 5 *A*, *B*, *C* and *D*.).—E. L., a 36-year-old foundry worker, first developed symptoms of pulmonary and spinal tuberculosis in 1930, but a diagnosis was not established until April, 1932. He was treated by bed rest, and an extrapleural pneumothorax on the right was performed Nov. 18, 1938. Following this he was admitted to a tuberculosis sanatorium in March, 1939, with a right extrapleural tuberculous empyema. This was treated conservatively by aspirations. Finally the area became opaque by roentgenograph, his sputum became negative, and he was discharged in September, 1941, as arrested. Also during this hospitalization a spine fusion was done, and his spinal disease was considered arrested. In December, 1942, he was readmitted because of a positive sputum and air and fluid in the extrapleural pocket. A broncho-extrapleural fistula was demonstrated by dye instillation and expectoration. By October, 1949, the space had again filled almost completely, but a small area of radiolucence still existed. From that time until the time of operation the scant sputum was negative for tubercle bacilli by concentration and culture. The left lung had been stable since 1945. Bronchoscopy on Jan. 17, 1950, revealed a normal tracheobronchial tree. As a preliminary procedure to right upper lobectomy and decortication of the extrapleural empyema sac, an upper stage tailoring thoracoplasty was performed on Jan. 19, 1950. Following this the broncho-extrapleural fistula reopened, and there was marked sputum production up to 14 ounces daily, positive for tubercle bacilli. Sputum production slowly decreased and the decortication and upper lobectomy were performed on Feb. 26, 1950. The postoperative course was completely uneventful, sputum immediately converted, and has been negative by concentration and culture to date.

This is a case of extrapleural pneumothorax complicated by extrapleural tuberculous empyema and a broncho-extrapleural fistula. Decortication of the extrapleural empyema sac, resection of the right upper lobe and associated fistula preceded by a tailoring thoracoplasty was definitive therapy.

CASE 6 (Fig. 6 *A*, *B* and *C*.).—C. W., a 48-year-old Negro laborer, whose tuberculosis was diagnosed in January, 1947. The tuberculosis was progressive, and in July, 1947, a left pneumothorax was induced, but was complicated by pleuropulmonary adhesions. Reexpansion was started in July, 1948, but was never complete, and a purulent tuberculous pleural effusion developed. This was repeatedly aspirated from 1948 until the time of

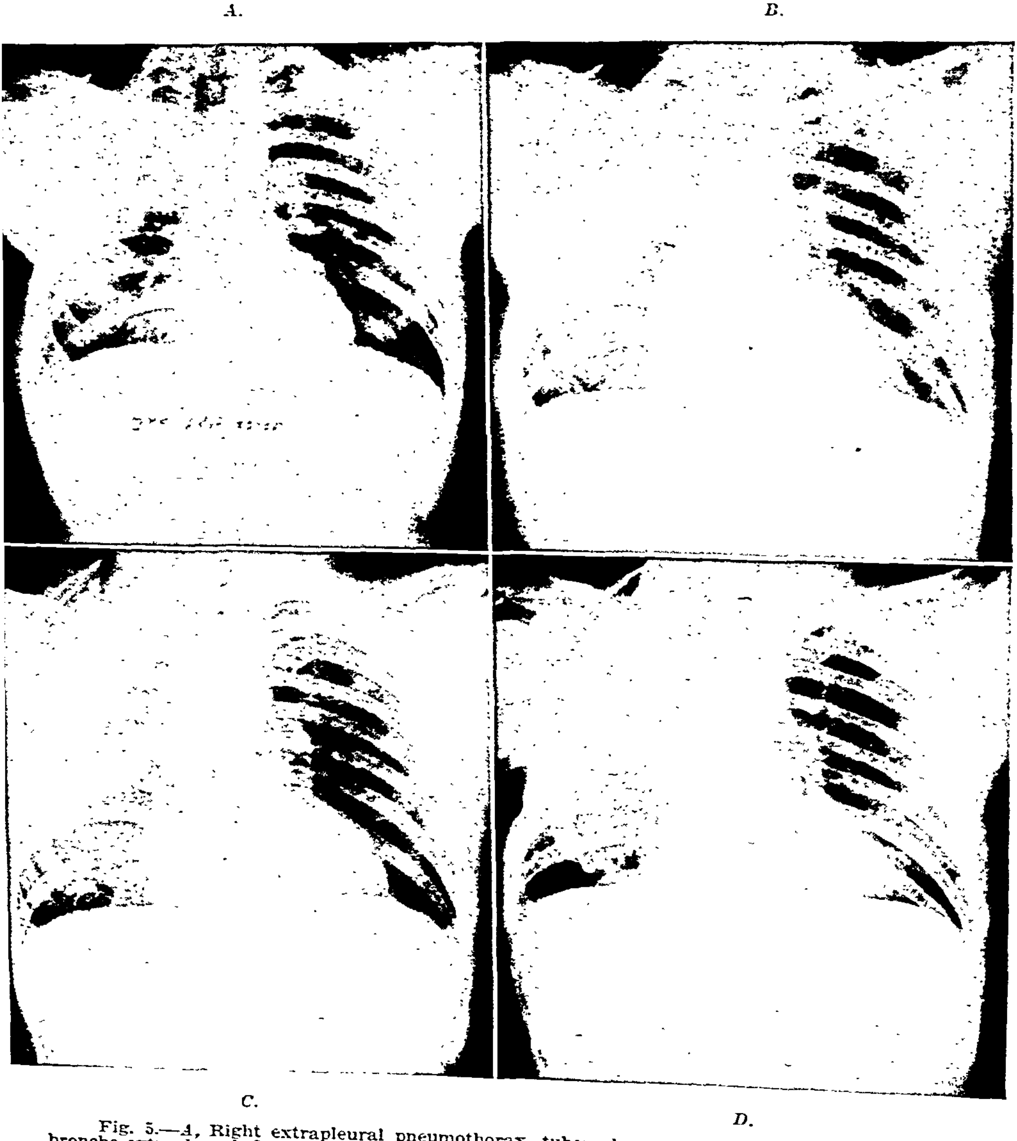


Fig. 5.—*A*, Right extrapleural pneumothorax. tuberculous empyema and right upper lobe broncho-extrapleural fistula; sputum. positive. *B*. Fistula closed and sputum negative temporarily. *C*, After tailoring thoracoplasty. fistula reopened and sputum positive. *D*. After decortication of extrapleural empyema and right upper lobectomy; sputum, negative.

cripple. The combination of left thoracoplasty and bilateral decortication has changed a previously hopeless situation to one where the patient is now contemplating gainful employment.

CASE 4 (*Fig. 4 A, B* and *C.*).—G. H., a 35-year-old man, a hoisting engineer, developed cough and sputum in 1948. Chest roentgenograph in July, 1948, disclosed cavitary tuberculosis on the right, and positive sputum was found. He was therefore hospitalized, and a right pneumothorax was begun in August, 1948, but was complicated by pleuropulmonary adhesions. In December, 1948, a complete closed pneumonolysis was performed. This was complicated by fluid development which persisted and was positive for tubercle bacilli. The upper lobe became almost 100 per cent collapsed. Because of these complicating factors, re-expansion was attempted, but was attended by reopening of the cavity in August, 1949. Finally in December, 1949, an upper stage of thoracoplasty on the right was done. On Jan. 24, 1950, the right middle and lower lobes were decorticated, and the right upper lobe was excised. Except for one isolated culture, this patient's sputum has been negative by all methods since February, 1950. The spheroidal lesions in the contralateral lung have remained unchanged and stable. Block resection of these lesions was under consideration, but, because of disciplinary reasons, this patient was discharged from the hospital in July, 1950, and since that date has been working full time, is asymptomatic, and continues to be sputum negative by all methods.

Here, despite adequate anatomical collapse by pneumothorax, parenchymal disease was uncontrolled, with patent cavity in the right upper lobe and smear positive sputum. This was further complicated by unexpandable lung and tuberculous empyema. Sputum conversion followed right upper lobectomy and decortication of the right middle and right lower lobes preceded by a tailoring thoracoplasty.

CASE 5 (Fig. 5 *A, B, C* and *D.*).—E. L., a 36-year-old foundry worker, first developed symptoms of pulmonary and spinal tuberculosis in 1930, but a diagnosis was not established until April, 1932. He was treated by bed rest, and an extrapleural pneumothorax on the right was performed Nov. 18, 1938. Following this he was admitted to a tuberculosis sanatorium in March, 1939, with a right extrapleural tuberculous empyema. This was treated conservatively by aspirations. Finally the area became opaque by roentgenograph, his sputum became negative, and he was discharged in September, 1941, as arrested. Also during this hospitalization a spine fusion was done, and his spinal disease was considered arrested. In December, 1942, he was readmitted because of a positive sputum and air and fluid in the extrapleural pocket. A broncho-extrapleural fistula was demonstrated by dye instillation and expectoration. By October, 1949, the space had again filled almost completely, but a small area of radiolucence still existed. From that time until the time of operation the scant sputum was negative for tubercle bacilli by concentration and culture. The left lung had been stable since 1945. Bronchoscopy on Jan. 17, 1950, revealed a normal tracheobronchial tree. As a preliminary procedure to right upper lobectomy and decortication of the extrapleural empyema sac, an upper stage tailoring thoracoplasty was performed on Jan. 19, 1950. Following this the broncho-extrapleural fistula reopened, and there was marked sputum production up to 14 ounces daily, positive for tubercle bacilli. Sputum production slowly decreased and the decortication and upper lobectomy were performed on Feb. 26, 1950. The postoperative course was completely uneventful, sputum immediately converted, and has been negative by concentration and culture to date.

This is a case of extrapleural pneumothorax complicated by extrapleural tuberculous empyema and a broncho-extrapleural fistula. Decortication of the extrapleural empyema sac, resection of the right upper lobe and associated fistula preceded by a tailoring thoracoplasty was definitive therapy.

Case 6 (Fig. 6 *A*, *B* and *C*.).—C. W., a 48-year-old Negro laborer, whose tuberculosis was diagnosed in January, 1947. The tuberculosis was progressive, and in July, 1947, a left pneumothorax was induced, but was complicated by pleuropulmonary adhesions. Reexpansion was started in July, 1948, but was never complete, and a purulent tuberculous pleural effusion developed. This was repeatedly aspirated from 1948 until the time of

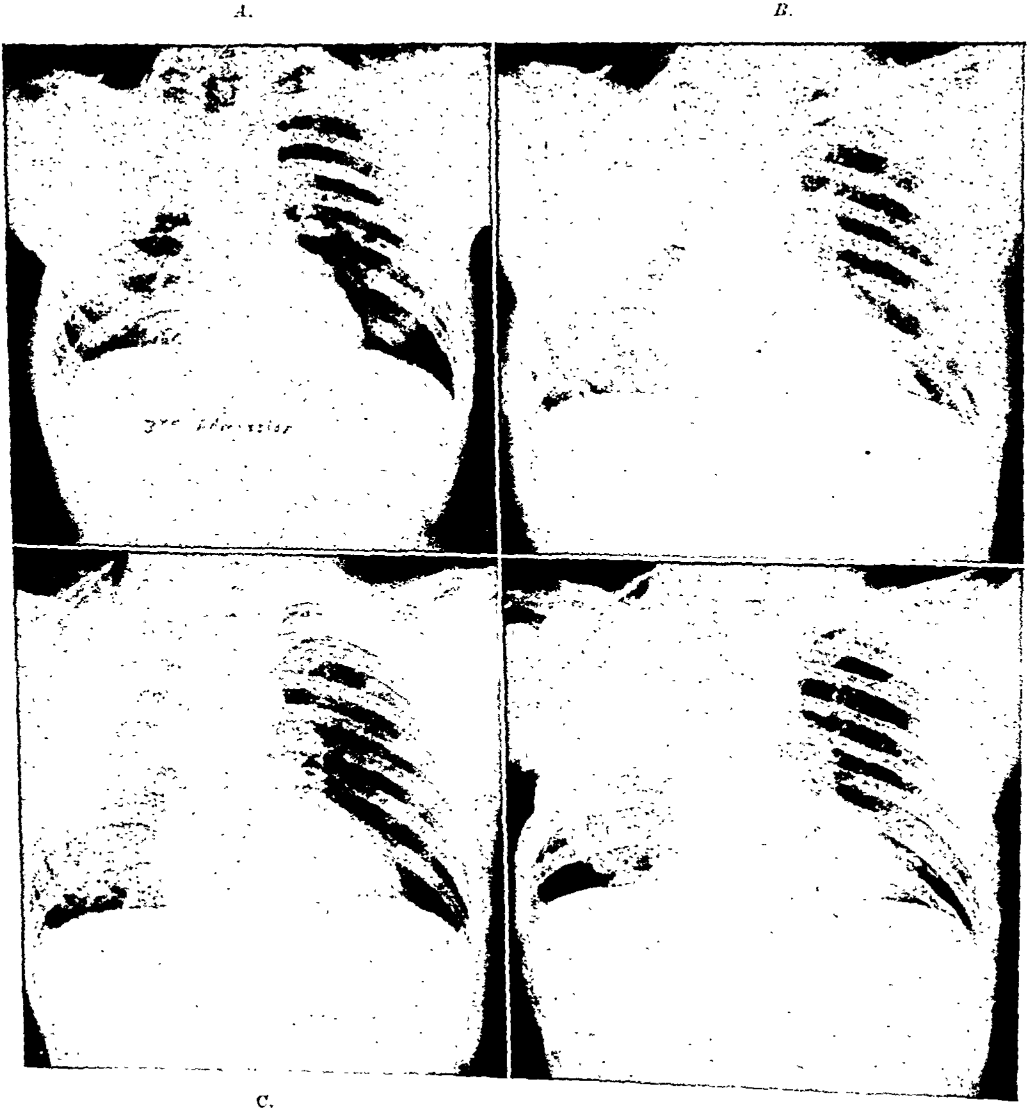


Fig. 5.—*A*, Right extrapleural pneumothorax, tuberculous empyema and right upper lobe broncho-extrapleural fistula; sputum, positive. *B*, Fistula closed and sputum negative temporarily. *C*, After tailoring thoracoplasty, fistula reopened and sputum positive. *D*, After decortication of extrapleural empyema and right upper lobectomy; sputum, negative.

surgery. Pneumonectomy was anticipated, but pre-empyema films were not complete enough to present a clear picture of the extent of involvement in the left lung. Therefore, it was decided to do a preliminary tailoring thoracoplasty to be followed by thoracotomy when decortication and pulmonary resection would be done. The extent of pulmonary resection would depend upon the amount of involvement found at operation. The preliminary thoracoplasty was done on Aug. 9, 1950, pneumonectomy and excision of empyema sac on Sept. 12, 1950, and a final stage of thoracoplasty on Oct. 19, 1950. Contralateral lung has remained stable throughout this procedure, sputum is now negative by all methods including culture, and he is on increasing activity.

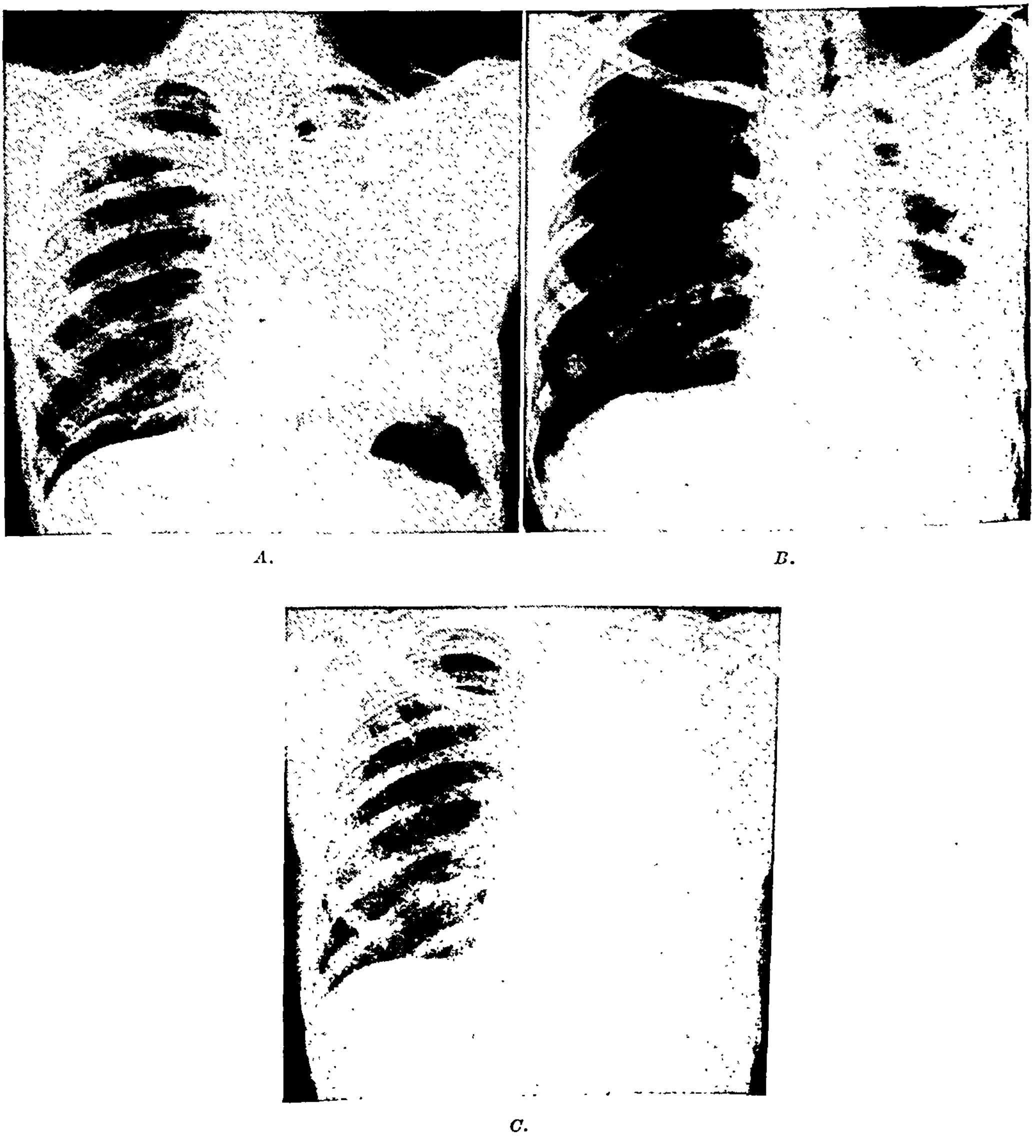


Fig. 6.—A, Extensive left lung tuberculosis with tuberculous empyema and unexpandable lung. B, After preliminary tailoring thoracoplasty. C, After left pneumonectomy, decortication of the empyema and a final stage of thoracoplasty; sputum, negative.

This represents the surgical program in a case with tuberculous empyema and uncontrolled parenchymal disease, the extent of which is difficult to determine by review of serial roentgenographs. Had a lobectomy and decortication been sufficient the surgical treatment would have been completed since preliminary tailoring thoracoplasty had been done. The finding of a destroyed lung required pneumonectomy and excision of the empyema. Obliteration of the pleural space was accomplished by a final stage of thoracoplasty.

CASE 7.—The case with complications of wound infection and residual empyema is a 39-year-old woman with total tuberculous empyema, cavitary tuberculosis of the left upper lobe, and endobronchial tuberculosis of the left lower lobe. She was known to have a bleeding tendency as demonstrated by the development of subcutaneous hematomas following needle aspirations and profuse bleeding during preliminary tailoring thoracoplasty. No cause of this hemorrhagic tendency could be determined. Despite this, the extensive tuberculous empyema demanded treatment. Decortication and pneumonectomy were started, but blood loss was so severe that operation had to be stopped after mobilization of the parietal portion of the empyema sac. The wound was packed, drained, and closed. Five days later the procedure was completed without difficulty. Despite completion of the thoracoplasty and pleural drainage, a small residual tuberculous empyema and moderate wound infection persists but is subsiding. The rarity of complications thus far in this series has been gratifying.

TABLE III A. RESULTS OF TREATMENT

Total number of cases		14
I. Successful pneumothorax—Decortication without resection		6
A. Without tailoring thoracoplasty	3	
B. With tailoring thoracoplasty	3	
II. Unsuccessful pneumothorax—Decortication with resection and tailoring thoracoplasty		6
A. Lobectomy	2	
B. Pneumonectomy	4	
III. Tuberculous pleurisy with effusion (spontaneous)		2
A. Unexpanded lung	1	
B. Empyema	1	

TABLE III B. RESULTS OF TREATMENT

Complications	
I. Wound infections	1
II. Reactivation or spread	0
III. Reversion of sputum	0
IV. Failure of re-expansion	0
V. Recurrence of empyema	1
Mortality	0

CONCLUSION

The mere demonstration of the presence of an unexpandable lung in the candidates for decortication is not a *sufficient criterion* for surgery. A thorough understanding of the natural history of pulmonary tuberculosis and a knowledge of the extent and activity of the existing disease are necessary as well as surgical skill. The finding of a positive sputum, even on culture only, becomes significant in planning the surgical program. The source of the bacilli should be determined if possible and then thoracoplasty and/or resection should be done in conjunction with decortication. Thus in the total group of cases presented for decortication the parenchymal status must be evaluated. For the cases with moderate prepneumothorax disease, complete re-expansion by simple decortication is desirable and adequate. In that group with more advanced parenchymal involvement permanent collapse of the diseased area is required. This is accomplished by preliminary tailoring thoracoplasty and then decortication of uninvolved lung. Finally, those cases unsuccessfully treated by pneumothorax, with persistent active disease and positive sputum, require resection and simultaneous decortication. Where more than segmental resection is done, preliminary tailoring thoracoplasty is also indicated to prevent overdistention of remaining lung and to insure obliteration of pleural space.

SUMMARY

The problem of the unexpandable lung after therapeutic pneumothorax and its complications is discussed.

A review of the surgical treatments of the unexpandable lung is presented.

Pathology of the unexpandable lung is briefly referred to.

The indications for decortication are discussed in detail. A reclassification of indications based on the status of the underlying lung as well as the pleural pathology is presented.

Concepts of treatment based on these indications are discussed. These include combinations of decortication, thoracoplasty and pulmonary resection.

A series of fourteen cases is reviewed and illustrative examples are presented.

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DECORTICATION PRECEDING THORACOPLASTY FOR THE ELIMINATION OF LONG-STANDING TUBERCULOUS EMPYEMA*

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THE elimination of chronic tuberculous empyema secondarily infected or not is a problem at any stage. The more long standing the empyema, the greater the problem. Granted that prevention is the best treatment, some cases are bound to occur, though possibly less frequently with improved management in general.

Early treatment by conservative methods may eliminate some empyemas, and early surgery will cure others. The long-standing cases with which we are mainly concerned are generally conceded to be amenable only to surgery. The generally accepted method has been by extensive thoracoplasty. This succeeds in some, but a high percentage, over half according to Cuthbert, results in residual basal empyema and chronic sinuses, even after the Shede type of procedures. It may be noted that Cuthbert states that half of these long-standing cases are not fit for any surgery, and this writer quotes a mortality of 50 per cent. It is appropriate also to draw attention to the high morbidity and prolonged treatment with multiple operations so common in this condition.

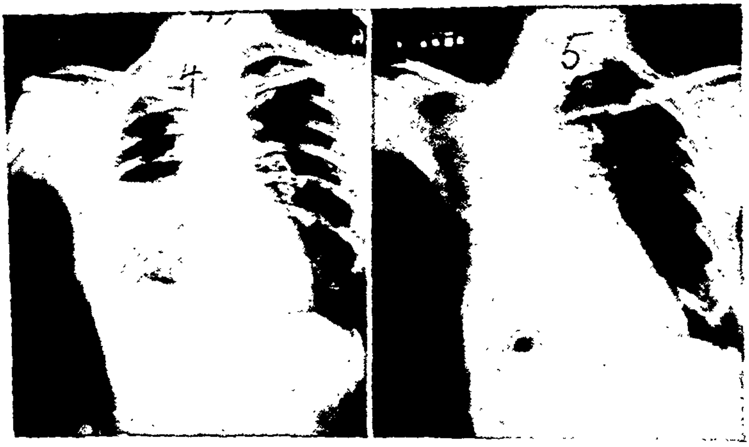
The decortication of chronic tuberculous empyema following thoracoplasty was practiced and discussed by the late Fraser B. Gurd before this Society in 1946. Subsequent contributions by Gordon and Welles, Weinberg and Davis, Mulvihill and Klopstock, and others have demonstrated that not only is this post-thoracoplasty decortication practicable, but that it could sometimes be carried out primarily and might even occasionally obviate thoracoplasty.

In reviewing our results with thoracoplasty in chronic empyema, it was noted that, other things being equal, surgery was most successful where the lower lobe had been well held out over the diaphragm by adhesions, and was only partially collapsed. In such cases (Fig. 1 *A*) a limited thoracoplasty combined with aspiration was generally adequate in achieving a cure of the empyema, leaving no sinuses or residual gutter pocket (Fig. 1 *B*). Such success was rarely the case with more complete lung collapse, when more extensive thoracoplasties regularly left draining sinuses from residual gutter empyemas (Fig. 2 *A*, 2 *B*, and 2 *C*). This regularly called for the mutilating Schede type of procedure with, all too frequently, further unsatisfactory results (Fig. 3 *A*, 3 *B*, and 3 *C*).

From Grace Dart Home Hospital, Royal Edward Laurentian Hospital and Queen Mary Veterans' Hospital.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

The satisfactory results in cases with the partially collapsed lung led us to attempt to imitate the natural intrathoracic conditions found in these cases in empyemas with more completely collapsed lung. This expansion was to be accomplished by a limited or lower lobe decortication followed by suction and later by aspiration. In other words, the plan was designed to coax the lower lobe out over the diaphragm where it could become adherent before thoracoplasty, whether the lung functioned or not. Thoracoplasty was to follow as soon as possible. This plan at the worst would preclude residual gutter empyema (Fig. 4 *A*), and ordinarily it would limit the subsequent thoracoplasty (Fig. 4 *B*), while at best it might make this operation unnecessary (Fig. 4 *C*). In Fig. 4 *A* a bronchogram indicates how even a relatively functionless lung can be made to suit the purpose of the plan as in Case 2, P. R. Fig. 4 *B* shows the limited thoracoplasty required in the same case, while Fig. 4 *C* illustrates the type of case in which subsequent thoracoplasty is not required.



A. *B.*

Fig. 1.—*A*. Chronic empyema following pneumothorax and pneumonolysis with partially collapsed right lower lobe naturally adherent well out over whole diaphragm despite exudate. *B*. Same case on discharge two months after completion of an eight-rib thoracoplasty, with only a brief aspiration period. Note absence of empyema. (Five-year follow-up shows patient healthy and working.)

It was early recognized that failure might result from certain pre-existing conditions, notably a bronchopleural fistula, extreme bronchial stenosis, or complete intrapulmonary fibrosis. These matters were given appropriate investigation in the cases encountered. It will be noted that only one of our patients had such a large fistula, and this case has so far been considered a failure. One patient had bronchial stenosis and was rejected. Several of the patients had extensive fibrosis, but this did not appear to defeat our purpose. These cases will be commented upon individually. We have thus perhaps been fortunate in our series, since at the outset no special selection was made, and most of the patients had been in and out of the hospital for many months, and the longest one six years.

The risk of expanding extensively diseased lobes has also been considered. After previewing all available old roentgenograms, none of our present series was considered as having too much involvement to be excluded from trial operation. However, such cases will be met with and each one must be dealt with on its merits. We have recently rejected two such candidates.

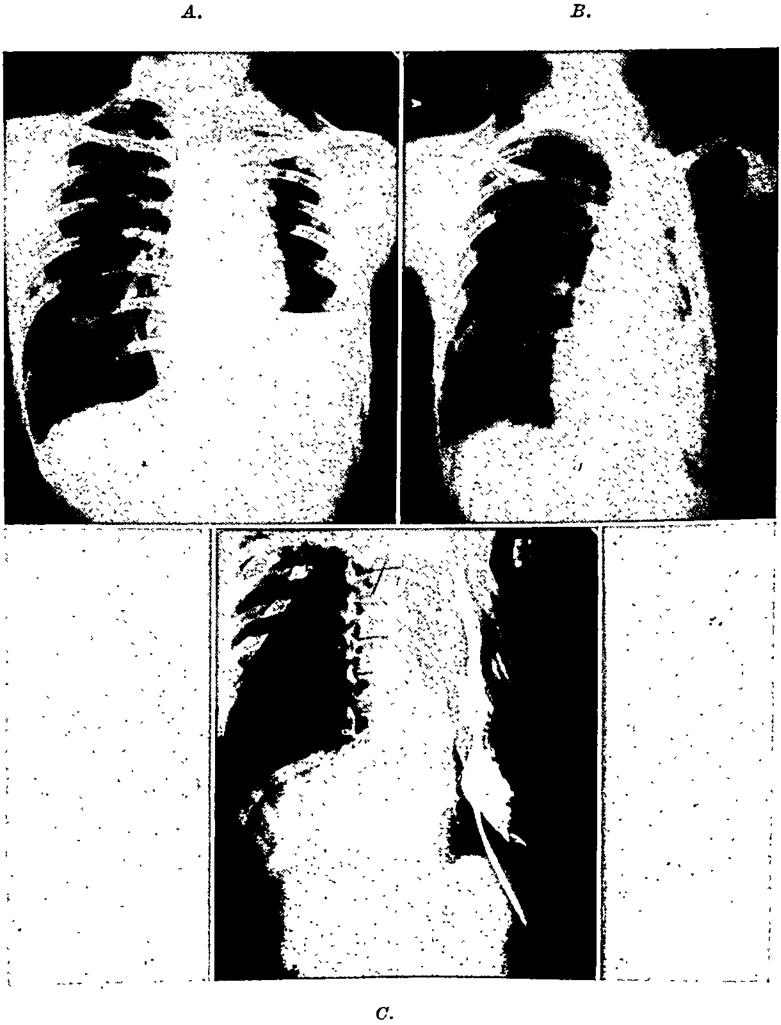


Fig. 2.—*A*, Chronic empyema following pneumothorax and pneumonolysis, with almost totally collapsed lung, nonadherent to the diaphragm, and showing some exudate. *B*, Same case three months after a standard nine-rib thoracoplasty showing residual gutter empyema and draining sinus. *C*, The residual cavity contains over 200 c.c. of Lipiodal.

The matter of functional restoration, or gain, in the expanded lung after decortication, has naturally also been investigated, though more or less a secondary consideration. It will be seen that the functional recovery varies widely in the individual case. Actually our results range from nil to 50 per cent normal, and are discussed individually. Functional recovery is of secondary interest to us in this paper, however, since we have been concerned primarily with the obliteration of, or prevention of, the almost inevitable residual gutter empyema and its disappointing sequelae, even following the Schede type of procedure.

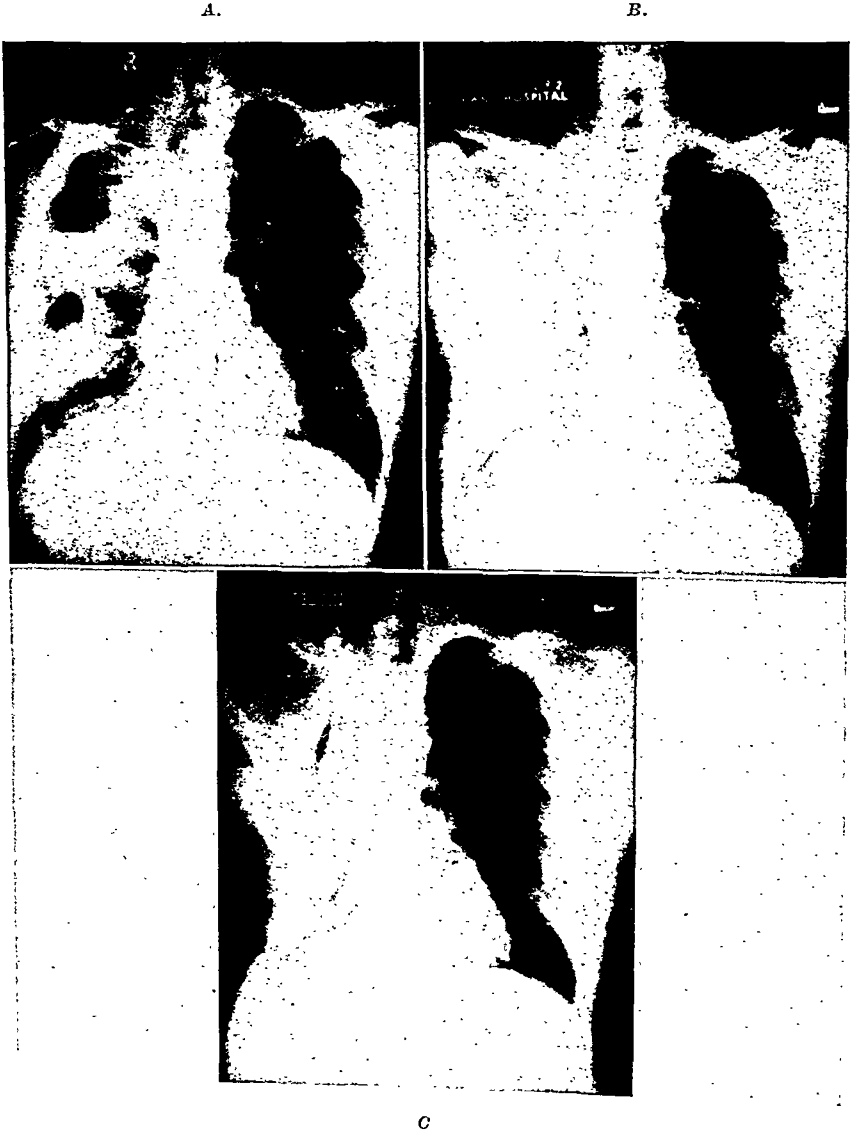


Fig. 3.—*A*, A case similar to that in Fig. 2. *B*, Same residual cavity six months after standard thoracoplasty. *C*, Following Shede operation, the gutter had finally dried up but only after two years.

Reports of ten cases are summarized and the results charted. It will be seen that a satisfactory and fairly rapid cure of empyema was effected in nine, and one has been a failure.

CASE REPORTS

Case 1.—P. R., man, aged 27 years, G.D.H.H. No. 4073. Disease was discovered as minimal in both apices in 1943, and the patient was admitted to a sanatorium. Right pneumothorax was begun in November, 1944. He left the sanatorium in September, 1945, and returned in June, 1946. Left pneumothorax was indicated and begun at once. Pneumonolysis was necessary, and this was shortly followed by effusion. Emypema was diagnosed later that year (1946) and was treated by aspiration and a long course of streptomycin. The patient was admitted to Grace Dart Home Hospital in June, 1948.

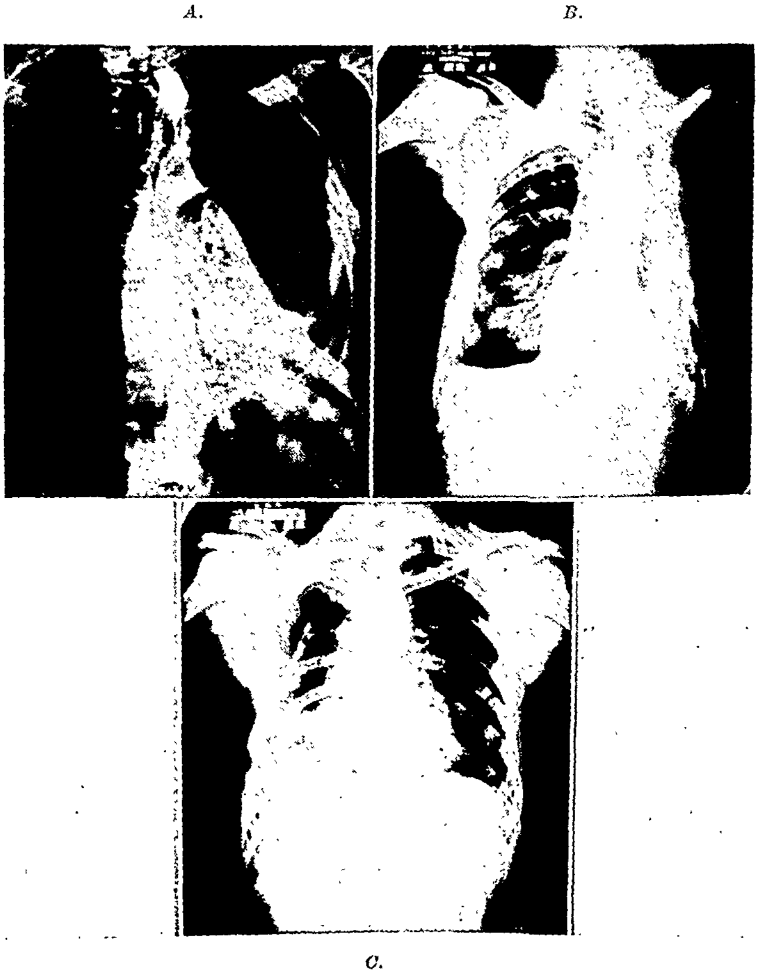


Fig. 4.—*A*, Bronchogram of the functionless left lung in Case 2, J. R., shows how even this type may be coaxed out to become adherent to diaphragm and serve our purpose. *B*, Following seven-rib thoracoplasty. *C*, Illustrates how decortication may even eliminate the necessity for thoracoplasty.

On examination the patient was febrile; sputum negative; weight 87 pounds. Chronic mixed left empyema with multiple draining aspiration sinuses. Pleural fluid showed acid-fast bacilli. The right side was considered stable. The blood picture was within relatively normal limits. The vital capacity was 1.2 liters. Bronchoscopy was negative. Bronchospirometry was attempted three times with unreliable results. There was no definite evidence of bronchopleural fistula.

On Feb. 1, 1949, decortication of the left lower lobe, lingula, and diaphragm was done with facility. Expansion of the lobe occurred with positive pressure on the table. Suction drainage with one 45F. tube was carried on for six days. Then the drain was removed and the wound sutured. Aspiration was continued, and the lower lobe remained well out on the diaphragm. Bronchospirometry showed minimal lung function (10 per cent). On March 29 a first-stage thoracoplasty was done, followed by second stage on May 10. Seven ribs in all

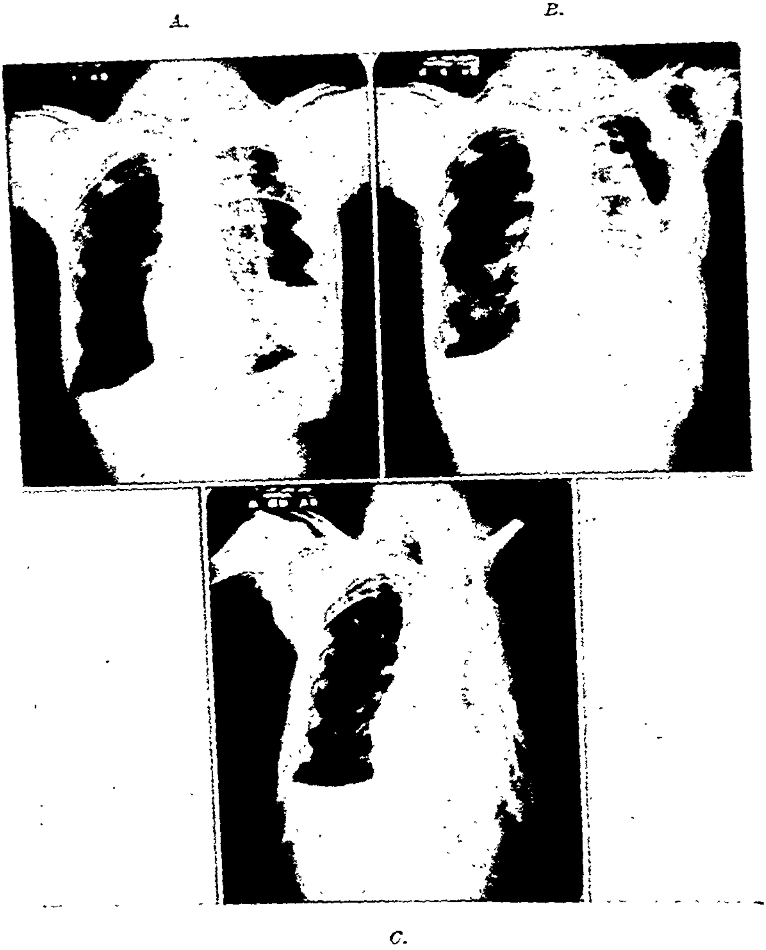


Fig. 5.—*A*, Case 1, P. R., showing collapsed lung in long-standing three and one-half year tuberculous empyema. Note thickened pleura. *B*, Expanded decorticated lower lobe before thoracoplasty. *C*, Postthoracoplasty result. There is a very minimal functional recovery.

were removed. The lung remained partly expanded. All aspiration ceased early in June because it became unnecessary. He was discharged on Sept. 16, 1949, weighing 95 pounds. The sputum has remained negative.

Follow-up in October, 1950: patient well and working (Figs. 5*A*, 5*B*, and 5*C*).

Case 2.—J. R., man, aged 24 years, G.D.H.H. No. 4809. Disease was discovered in 1944, following repeated hemoptyses. Admitted to sanatorium, and a left-sided cavitary lesion was diagnosed. Pneumothorax was begun early in 1943, and was followed shortly by pneumonolysis. The pneumothorax was abandoned in 1943 because of effusion. Empyema was diagnosed in 1944. He was self discharged in 1946. He was readmitted to a sanatorium in February, 1948, and transferred to the Grace Dart Home Hospital the same month, and streptomycin treatment was added to aspiration.

On examination the patient was febrile. There was a left-sided chronic tuberculous empyema with a positive culture. There was a small tuberculous puncture abscess of the left axilla. The right chest was considered stable. Sputum was negative; weight 92 pounds; the blood picture showed moderate anemia; the vital capacity was 1.9 liters. Bronchoscopy showed early bronchitis in the left upper lobe bronchus. Bronchospirometry showed no oxygenation from the left lung. There was no definite evidence of a bronchopleural fistula.

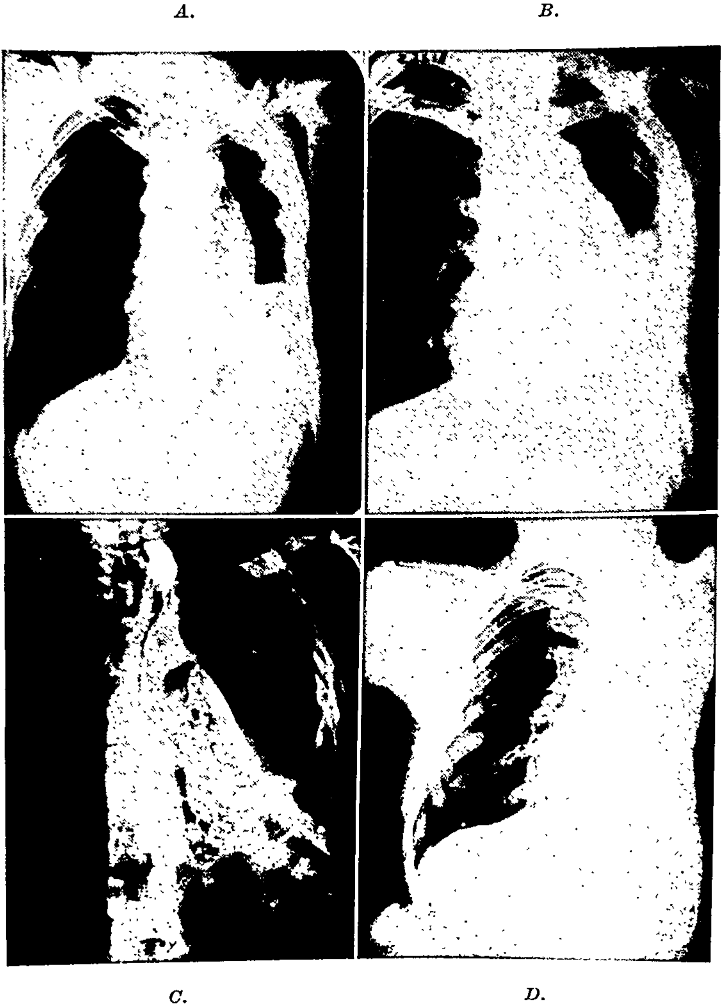


Fig. 6.—*A*, Case 2, J. R., shows collapsed lung in a six-year tuberculous empyema. *B*, Extent of expansion after decortication with minimal functional recovery. *C*, Bronchogram of same case showing how the lung has crept out over the diaphragm. *D*, Final result with minimal function.

On March 11 an incision and drainage of the axillary abscess was done. On March 16 decortication of the left lower lobe, lingula, and diaphragm was carried out quite easily. The lung did not expand under positive pressure on the table. Drainage of the left chest by one 45F. tube with suction was continued for one week. Then the tube was withdrawn and the wound sutured. Aspiration was continued. The fibrosed lung remained well out over the diaphragm, as is well shown by bronchogram roentgenogram. On April 4, a bronchospirometry was done, showing no oxygenation of the left lung. On April 20 and 30 a limited seven-rib two-stage thoracoplasty was carried out. Aspiration ceased in May. He was discharged from the hospital to home on July 5, 1949.

Follow-up in October, 1950, found the sputum negative and the patient well and working (Figs. 6*A*, 6*B*, 6*C*, and 6*D*).

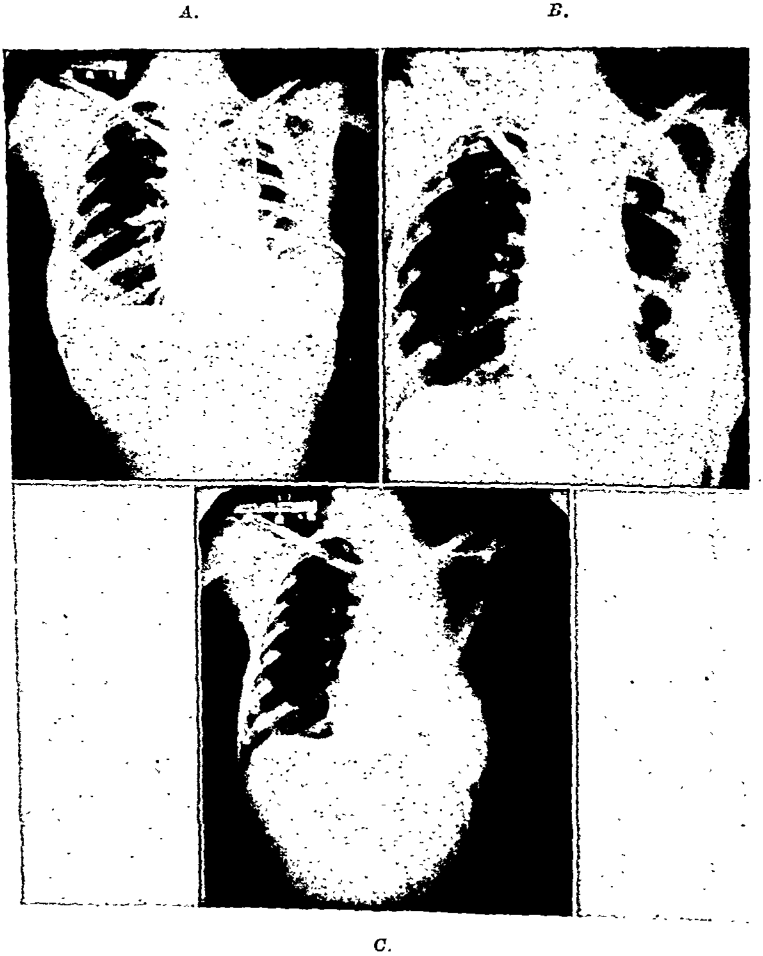


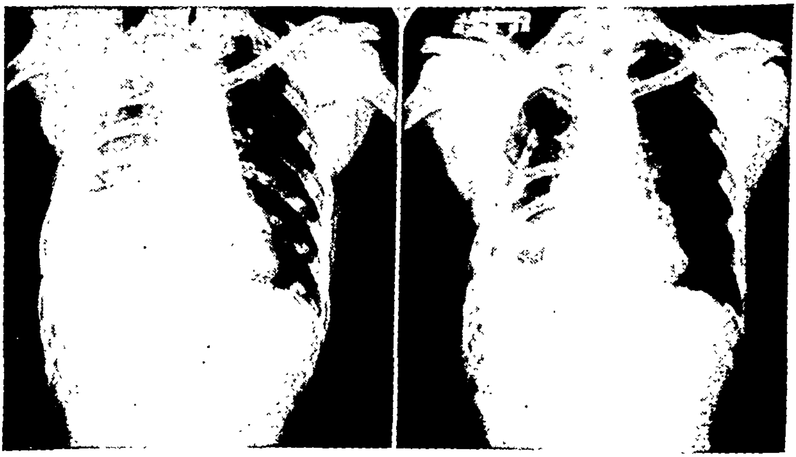
Fig. 7.—*A*, Case 3, T. N., shows collapsed lung with drainage tube. *B*, Functionless lower lobe just filling the gutter area. *C*, Final result after thoracoplasty.

Case 3.—T. N., woman, aged 31 years, G.D.H.H. No. 4994. Disease was first recognized in 1943. She entered a sanatorium in 1944, where the left lung was found to have a cavitary lesion. Left pneumothorax was begun at once and continued for two and one-half years. It was abandoned because of effusion in 1946. At that time the right side showed some infiltration. In 1948 she was discharged home because her sputum was negative. She was readmitted to sanatorium with empyema in October, 1949, and treated by aspiration and *streptomycin. In February, 1950, she was transferred to the Grace Dart Home Hospital for* surgery, with a drainage tube in her left chest.

On examination the patient was febrile; weight 90 pounds. She showed bilateral pulmonary tuberculosis, and the right lung was considered to be stable. There was a left-sided total empyema which contained Staphylococci and *B. coli,* but was negative for tubercle bacilli. There was a small drainage tube in situ. She had a small amount of negative sputum. The blood picture showed moderate anemia. The vital capacity was 1.1 liters. Bronchoscopy was negative. Bronchospirometry was unsatisfactory. There was no definite gross bronchopleural fistula.

On March 7, 1950, decortication of the left lower lobe, lingula, and diaphragm, and the lower parietal pleura was done. The lung expanded only slightly under positive pressure on *the operating table. Drainage with appropriate suction by two 45F. tubes was carried on for* six days, and then the wounds were sutured. Aspiration was continued. The lung remained partly expanded and adherent to the diaphragm. On April 5 and 25, a limited seven-rib two-stage thoracoplasty was done. Aspiration ceased in May. She was discharged on Oct. 6, 1950, when her weight was 96 pounds.

Follow-up in February, 1951, showed the patient at home and well (Figs. *7A*, *7B*, *7C*, and *7D*).



A. *B.*

Fig. 8.—*A*, Case 4, L. P., shows the empyema with only partial collapse of stable lung. *B*, Final result of decortication, requiring no thoracoplasty and rendering a 30 per cent lung function.

CASE 4.—L. P., man, aged 27 years, G.D.H.H. No. 4771. Disease was discovered in July, 1946, following pleurisy with effusion on the right side. He was admitted to a sanatorium in 1946, then discharged home. He was readmitted to a sanatorium in 1947 for thoracotomy with tube drainage for chronic empyema of mixed type. He was then transferred to the Grace Dart Home Hospital in September, 1948.

On examination he was febrile. The left lung was clear. The right side showed a chronic empyema with small draining sinus in the lower axilla. The upper lobe was partly expanded and functioning. Cultures from empyema showed *M. tuberculosis*, Staphylococci, and other organisms. His weight was 110 pounds. The vital capacity was 1.3 liters. Bronchoscopy was negative. Bronchospirometry showed 20 per cent function on the affected side. There wes no evidence of bronchopleural fistula.

On Jan. 11, 1949, decortication of the right lower and part of the upper and middle lobes was carried out. Suction drainage was continued for five days with a 45F. tube. Drainage wound was then sutured and aspiration continued. The lung which had never been completely collapsed re-expanded under aspiration, and showed no active disease. Thoracoplasty was not done. Intermittent aspiration of a localized pocket of thin serum, negative for organisms, was continued until March. Bronchospirometry in August, 1949, showed increased function of the right lung estimated to be 30 per cent. He was discharged on Oct. 17, 1949. His weight at this time was 115 pounds.

In October, 1950, he was seen at the Clinic. He was well and working (Figs. 8*A* and 8*B*).

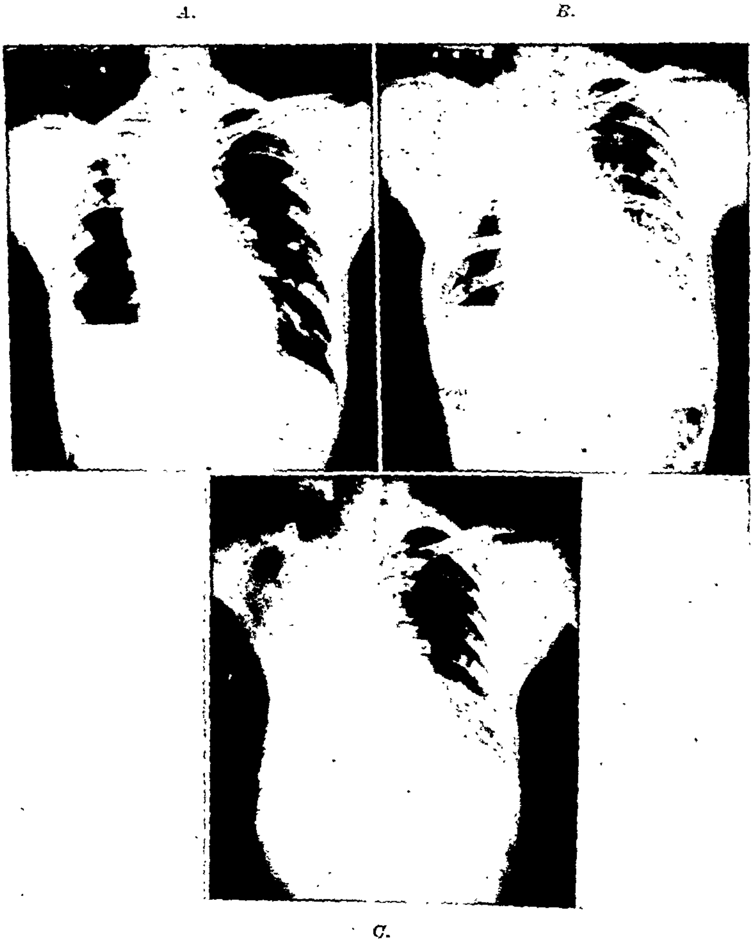


Fig. 9.—*A*, Case 5, R. D., shows empyema with total lung collapse. *B*, Condition following two-stage six-rib thoracoplasty. *C*, Result following completion of thoracoplasty and simultaneous decortication.

Case 5.—R. D., man, aged 26 years, G.D.H.H. No. 4969. The disease was recognized in October, 1948. The patient was admitted to a sanatorium in October. He developed a right-sided spontaneous pneumothorax in January, 1949. Effusion followed almost at once, and this after six months gradually became purulent. He was transferred to the Grace Dart Home Hospital on Dec. 7, 1949, for surgery after nine months' treatment by aspiration and streptomycin.

On examination he was febrile. Roentgenogram revealed far-advanced bilateral disease. He had positive sputum. The empyema contents revealed a mixed infection. A two-stage six-rib thoracoplasty was done in February and March, 1950. A residual gutter empyema cavity remained. Small bronchopleural fistula was suspected.

On March 29 a third-stage thoracoplasty was performed removing ribs 7, 8, and 9; and decortication of the residual empyema and freeing of the diaphragm was done. There was minimal expansion of the lobe on the operating table. Drainage was accomplished through the eighth rib bed with a 45F. suction tube and carried on for six days, and then closed and followed by aspiration. This was followed by a phrenic crush on the right. Aspiration was no longer necessary after May, and he was then discharged to a sanatorium the same month.

He is now well and working (Figs. 9*A*, 9*B*, and 9*C*).

Case 6.—A. M., man, aged 20 years, R.E.L.H. The disease was first recognized in January, 1948, as a cavitary lesion of the left upper lobe and infiltration of the lower lobe. The patient was admitted to a sanatorium, and a left pneumothorax was begun in April, 1948. He had a pneumonolysis in July, 1948, and again in January, 1949. This was followed by an effusion which was treated by aspiration. Tubercle bacilli were found in the effusion in September, 1949. He was transferred to the Royal Edward Laurentian Hospital for Surgery in November, 1949.

On examination the patient was febrile, with a negative sputum. There was a left-sided chronic tuberculous empyema with a positive culture. The right lung showed infiltration but was stable. There was no bronchopleural fistula. Bronchoscopy was negative. Bronchospirometry showed 80 per cent of function on the right and 20 per cent on the left side. The vital capacity was 3.2 liters.

On Jan. 30, 1950, decortication of the left lower and part of the upper lobe was done. Expansion on the table was immediate and good. Suction drainage was done for five days, and then the wound was sutured. The lung continued to expand and radiologically was stable enough so that thoracoplasty was not done. He was returned to the sanatorium after six months and has remained there since. The functional recovery of the expanded lung has been estimated by bronchospirometry to be 50 per cent (Figs. 10*A*, 10*B*, and 10*C*).

Case 7.—D. M., man, aged 51 years, R.E.L.H. The disease was first recognized in January, 1950, and the patient was admitted to a sanatorium with a positive sputum. Pneumothorax was begun in April. In May, 1950, a spontaneous pneumothorax occurred following a refill. Effusion followed shortly and was treated by aspiration and streptomycin. In June, 1950, the tubercle bacilli were found in the thickening effusion. He was transferred to the Royal Edward Laurentian Hospital for surgery.

Examination on admission showed the patient to be febrile, and a positive sputum. In the background there was infiltration of both lungs with an upper cavitary lesion on the right. His weight was 140 pounds; vital capacity 2.6 liters; bronchoscopy showed atelectasis but no stenosis. There was no evidence of a bronchopleural fistula. Bronchospirometry was not done.

On June 12 a lower lobe decortication was done with good expansion on the table under positive pressure. Within a week suction drainage was discontinued, and the wound was closed. Aspiration was continued. In September and October he had a two-stage seven-rib thoracoplasty done, which cured the empyema and converted him.

He is presently in the sanatorium and remains well.

Case 8.—G. D., man, aged 25 years, Q.M.V.H. The disease was found in June, 1948, and he was admitted to a sanatorium where a left pneumothorax was induced. In December, 1948, effusion became prominent, and six months later *M. tuberculosis* were found. He was treated by aspiration and streptomycin without success. He was transferred to the Queen Mary Veterans' Hospital in October, 1949.

Examination on admission showed the patient to be febrile. The sputum was negative. He had a left-sided tuberculous empyema. The contralateral side was clear. Bronchoscopy was negative. No bronchopleural fistula was present. The vital capacity was 1.6 liters. Bronchospirometry on the left was 1 per cent.

In December, 1949, lower lobe decortication was done with immediate expansion, and this was shortly followed by a two-stage seven-rib thoracoplasty. The subsequent course was uneventful, and after one year he is well in the sanatorium.

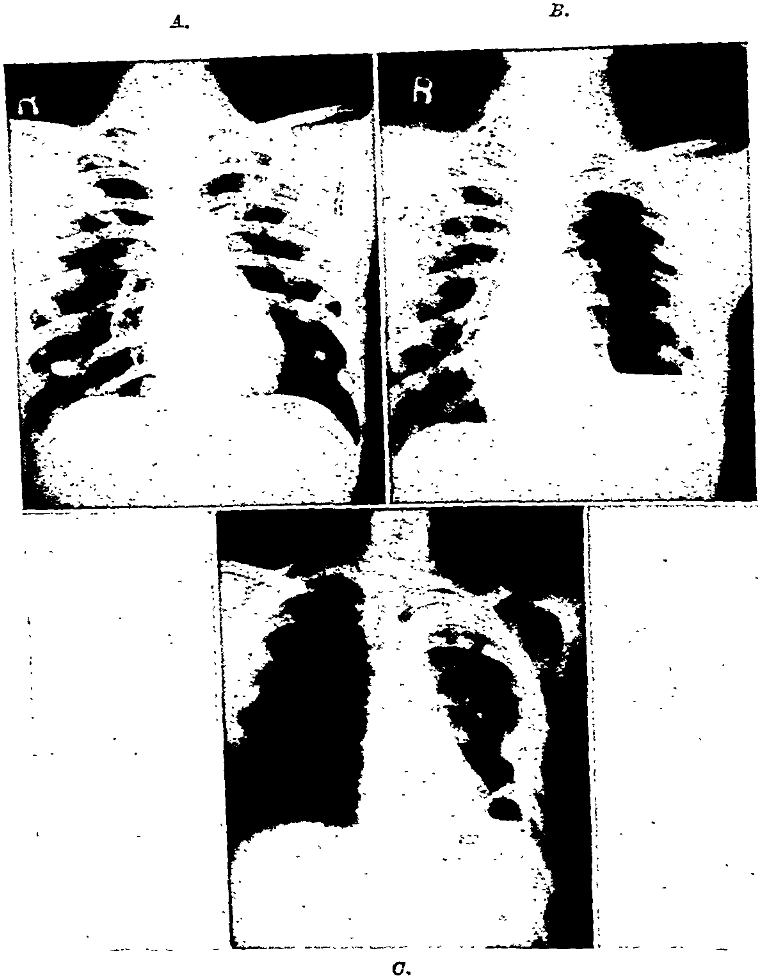


Fig. 10.—*A*, Case 6, A. M., shows original condition of lungs. *B*, Empyema. *C*, Stable, expanded lung with 50 per cent functional recovery requiring no thoracoplasty.

CASE 9.—I. L., man, aged 35 years, Q.M.V.H. The onset of disease was in 1947, when he was admitted to a sanatorium where a right pneumothorax was shortly begun. The effusion became empyema in 1948, and he received conservative treatment for the next year. In 1949 he was transferred to the Queen Mary Veterans' Hospital for surgery.

On admission the patient was febrile, and had a positive sputum. There was a right-sided tuberculous empyema and the contralateral side showed infiltration. Bronchoscopy showed no definite bronchial disease, and the bronchopleural fistula was only recognized later. Vital capacity was 2 liters.

TABLE I. ANALYSIS OF CASES

CASE NO.	1-P.R.	2-T.R.	3-T.N.	4-L.P.	5-R.D.	6-A.M.	7-D.M.	8-G.D.	9-I.L.	10-G.B.
Type of empyema	mixed	Tbc.	mixed	mixed	mixed	Tbc.	mixed	Tbc.	Tbc.	Tbc.
Condition underlying lung originally	upper cavity	upper cavity lower infilt.	upper cavity	infilt. all zones	extens. cavity	upper cavity	upper cavity lower infilt.	upper cavity	upper cavity	upper infilt.
Contralateral lung	stable	clear	stable	clear	stable	infilt.	stable	stable	infilt.	stable
Sputum	neg.	neg.	neg.	neg.	pos.	neg.	pos.	neg.	pos.	neg.
Bronchopleural fistula	0	0	0	0	+	0	0	0	+	0
Bronchoscopy	neg.	neg.	neg.	neg.	neg.	neg.	neg.	neg.	pos.	neg.
Vital capacity	1.2 L.	1.9 L.	1.1 L.	1.3 L.	2.2 L.	3.2 L.	2.6 L.	1.6 L.	2 L.	3 L.
Bronchospirometry	unrel.	no func.	unrel.	20%	not done	20%	not done	1%	not done	not done
Extent of fibrosis	++	+++	++++	+	+++	+	++	++++	++++	+
Age	27	24	31	27	26	20	51	25	35	37
Sex	M	M	F	M	M	M	M	M	M	F

TABLE II. ANALYSIS OF CASES

CASE NO.	1-P.R.	2-J.R.	3-T.N.	4-L.P.	5-R.D.	6-A.M.	7-D.M.	8-G.D.	9-I.L.	10-G.B.
Extent of decortication	L.L.L.	L.L.L.	L.L.L.	R.L.L. & MID.	R.L.L.	L.L.L. PT.UPP.	R.L.L.	L.L.L.	R.L.L.	R.L.L. MID.
Expansion of lung	immed. partial	min.	immed. partial	immed. good	min.	immed. good	immed. good	immed. good	min.	immed. good
Extent of thoracoplasty	7 ribs	7 ribs	7 ribs	none	9 ribs	none	7 ribs	7 ribs	8 ribs	none
Functional recovery	min. 10%	nil.	min.	30% of normal	nil.	50% of normal	min.	min. 1-5%	nil.	fair
Period effusion to empyema	6 mo.	18 mo.	18 mo.	1 yr.	9 mo.	1 yr.	2 mo.	6 mo.	1 yr.	6 mo.
Length conservative treatment	2½ yr.	4½ yr.	6 mo.	3 yr.	9 mo.	1 yr.	2 mo.	1 yr.	1 yr.	6 mo.
Total pre-op. time	3 yr.	6 yr.	2 yr.	4 yr.	18 mo.	2 yr.	4 mo.	18 mo.	2 yr.	1 yr.
Period oper. to discharge	6 mo.	3 mo.	6 mo.	9 mo.	3 mo.	3 mo.	3 mo.	3 mo.	not disch.	6 mo.
Follow-up	18 mo.	2 yr.	1 yr.	2 yr.	1 yr.	1 yr.	9 mo.	1 yr.	1 yr.	2 yr.
Result	satisfactory	satisfactory	satisfactory	satisfactory	satisfactory convert.	satisfactory	satisfactory convert.	satisfactory	failure	satisfactory

In June, 1949, lower lobe decortication was carried out with moderate expansion. Shortly after the operation the newly expanded lung collapsed, however. The empyema became secondarily infected, and the bronchopleural fistula was recognized. Thoracoplasty was done in three stages, but the patient has a persistent gutter cavity of mixed empyema and chronic sinus.

CASE 10.—G. B., woman, aged 37 years, Q.M.V.H. The patient's disease was found in 1948, and she received a right pneumothorax in the sanatorium. Effusion developed shortly afterward and this became recognizable empyema in May, 1949. For the next six months conservative measures were adhered to, then she was transferred to the Queen Mary Veterans' Hospital for surgery.

Examination on admission showed that the patient was febrile, and she had a negative sputum. There was right-sided tuberculous empyema and minimal areas of stable infiltration in the opposite lung. Bronchoscopy was negative, and there was no bronchopleural fistula. Vital capacity was 3 liters.

In September, 1949, decortication of the right lung was done with immediate good expansion. No thoracoplasty was needed as the underlying expanded lung was stabilized. The functional recovery has been only fair, however, but not accurately estimated.

COMMENT

Although the technique used and management following operation are now generally known and need not be recounted, a few comments are indicated.

Generally speaking, the visceral peel has been developed and removed with no great difficulty, leaving islands wherever very adherent to underlying visceral pleura or lung. In only two cases was the visceral peel so densely adherent as to make removal tedious, and these were cases with extensive pulmonary fibrosis. The diaphragm has been freed and some respiratory movement obtained in each instance, otherwise the amount of parietal decortication has been variable, and tended to be minimal. The upper lobe has been partly decorticated in three instances where more complete expansion was anticipated and considered desirable. In these instances considerable functional lung was restored and thoracoplasty obviated. Tears in the visceral pleura, where appreciable, have been sutured at once to minimize air escape. This has been variable and on the whole not remarkable. Fibrin foam has been used at times. Using endotracheal anesthesia, positive pressure expansion of the decorticated lung has been attempted in every instance with varying results, as shown on the summary chart. When expansion has occurred it has not been subsequently lost except in the one failure. Postoperative suction drainage has been accomplished with one 45F. tube and maintained for not more than a week in any case. The suction pressure varied from 10 to 20 cm. of water. After removal the drainage wounds were deliberately sutured, and aspiration substituted. Persistent sinus tracts occurred in our one failure. The aspirated fluid has never become purulent in any case but the failure. Streptomycin and sulfonamides have been used topically.

In Case 5 thoracoplasty was begun before the decortication which was carried out actually at the third stage. The variation in procedure was adopted in an effort to offset the existing bronchopleural fistula. Though successful, it was found technically more difficult than operation upon the uncollapsed thorax. In this case a phrenic crush was added, but not in the others.

Our results so far have been encouragingly successful, as will be seen from the preceding case reports given with minimal detail. For easier study we have added charts embodying the principal data. It may be noted that there have been no deaths, and morbidity has been remarkably light. As a result of this, the plan is calculated to cut the patient's hospitalization notably.

We wish to emphasize the time element in these results—most of the patients have been lying around sanatoria or hospitals for years.

SUMMARY

1. The unsatisfactory and mutilating results of the past surgery for chronic tuberculous empyema, including Schede type of operations, has engendered the plan to decorticate before thoracoplasty. This plan is particularly useful for the more completely collapsed lung, and decortication should include at least the lower lobe and diaphragm. More extensive visceral and parietal clearing should be done as indicated.

2. This plan was suggested by the observation that in those chronic empyemas with the lower lobe adherent to the diaphragm, a limited thoracoplasty is generally successful in preventing residual gutter empyema and draining sinuses.

3. The results of the plan have been generally satisfactory in our small series, even when the empyema was as long standing as six years. By satisfactory we simply mean the elimination of the empyema.

4. This result is our prime consideration and may occur with or without more or less functional improvement in the collapsed lung. Such a gain in function has occurred in some cases and was well marked in two. This accrued benefit makes the decortication plan the more valuable.

5. The fairly rapid recovery and early discharge from hospital, even in very long-standing cases, are facts which may well be stressed.

6. There have been no deaths in our series, and morbidity following operation has not been noteworthy.

7. Our one failure had an appreciable bronchopleural fistula. This may be considered as a possible contraindication, at least in the large ones, though we would like more experience. It is possible that means may be found to circumvent this difficulty.

8. Another more definite contraindication is underlying active pulmonary disease forbidding lung expansion.

9. Extensive pulmonary fibrosis is not a contraindication, since functional recovery is not a prime consideration, as has been demonstrated.

10. This argument may also apply to bronchial stenosis, though it is by no means clear.

11. Most of our successful cases have been followed for well over a year with satisfactory results.

12. The successful application of decortication procedures before, during, and after thoracoplasty gives hope that these procedures will largely eliminate the more mutilating operations.

13. Finally, a plea should be made for not delaying the surgery once the empyema is established. This plea is underlined by the greater likelihood of achieving maximum functional recovery, even making thoracoplasty unnecessary in a fair percentage of cases.

Of the ten cases included in this report five are from the Grace Dart Home Hospital, two from the Royal Edward Laurentian Hospital, and three from The Queen Mary Veteran's Hospital, all of Montreal. We are indebted to Dr. C. A. MacIntosh for permission to include his case reports, also to Dr. E. A. MacNaughton for his interest and slides.

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DISCUSSION ON "SIMULTANEOUS DECORTICATION AND RESECTION IN UNSUCCESSFUL PNEUMOTHORAX," BY DR. ARNOLD O. RILEY AND DR. VICTOR H. KAUNITZ; AND "DECORTICATION PRECEDING THORACOPLASTY FOR THE ELIMINATION OF LONG-STANDING TUBERCULOUS EMPYEMA," BY DR. F. DOUGLAS ACKMAN, AND DR. PATRICK MADORE.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—We are indebted to Dr. Riley and Dr. Kaunitz, and to Dr. Ackman as well. Essentially, the indications for decortication given by Dr. Riley and Dr. Kaunitz are in agreement with ours, and are those that the Committee on Surgical Therapy of the American College of Chest Physicians, of which I was a member and Dr. Abbott was chairman, presented at San Francisco last year. Certainly the indications are widening, as you can well see.

As to the group that requires resection, I think it might be mentioned that by and large those patients having extensive disease, and thus not suitable for decortication, have probably been poorly selected for pneumothorax in the first place. When we do resections on such patients we are, in essence, substituting the correct therapy that should have been applied in the first place. On the other hand, the case that has been well selected for pneumothorax will probably be ideal for decortication.

As to surgical procedure, we are in entire agreement that the parietal peel should be removed in all cases. We disagree, however, in the resection of the rib. We feel that an intercostal incision in decortication, even when the ribs are almost overlapped, is quite feasible with the use of the Inge bone spreader, an instrument we mentioned in a recent publication. With this instrument the ribs can be spread apart, the intercostal space divided accurately, and the parietal peel stripped away before it is incised. Then with the use of an ordinary rib spreader, gradually tightened, an adequate exposure can be obtained without division of any ribs whatever. For several years we have advocated this incision for all intrathoracic work, in fact, and we think that postoperative function of the chest wall is far better if there is minimal interference with both musculature and rib cage.

DR. KAUNITZ, (closing): Dr. Riley and I want to thank the Association very much for the privilege of addressing this meeting.

For the reasons Dr. Chamberlain gave earlier this morning, we have felt that thoracoplasty as a preliminary procedure should be done in a separate stage. We realize that other surgeons are using the same procedures we have indicated, but we felt that it was worth while to describe what we consider a logical method of handling these somewhat complicated situations.

SUBSCAPULAR PARAFFIN PACK AS A SUPPLEMENT TO THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

W. E. Adams, M.D.,* William M. Lees, M.D.,** and
James M. Fritz, M.D.* (by invitation)
Chicago, Ill.

The modern type of paravertebral thoracoplasty is the operation of choice in a large percentage of patients with pulmonary tuberculosis where surgical therapy is indicated. This operation has withstood the test of time and yields a high percentage of satisfactory results. Concomitantly, when it is carried out in an adequate number of stages, the risk of the procedure is minimal and the complications few.[1]

In spite of the contributions this procedure has made in the management of pulmonary tuberculosis, it has several disadvantages which leave room for improvement. Foremost among these is the fact that a significant percentage of patients are left with a persistently positive sputum.[2] Thus, further surgery is required either in the form of collapse or resection therapy. This means that the morbidity, as well as the risk of therapy, is increased. A second factor is the necessity for the employment of a multiple stage procedure in order to safeguard against paradoxical motion with spread of the disease to other parts of the lung. Of less importance is a third factor; i.e., the resultant deformity due to scoliosis and a sinking in of the scapula and subclavicular area.

All of these features must be considered when deciding in favor of therapy by thoracoplasty or by resection of the involved area. Although in recent years the mortality of operation and incidence of complications associated with the resection procedure have decreased considerably due to the use of chemotherapy, they still remain a deterrent factor in some types of lesions.

In an effort to improve the benefits of surgical collapse from time to time, a number of procedures have been employed with variable degrees of success. Some of these have been extrapleural in type, others have been subcostal interventions: Extrapleural pneumonolysis and pneumothorax (Tuffier[3]), with gauze pack (Schlange[4]), with fat (Tuffier[5]), with muscle (Alexander[6]), with rubber sheeting (Lilienthal[7]), with paraffin (Baer[8]) and with plastic material (Wilson[9]); supraperiosteal and subcostal pneumonolysis with rubber bag (Sherer[10]), gauze pack (Lilienthal[7]), muscle (Alexander[5]), paraffin (Eloesser[11]) and plastic material (Lucas[12]). When their employment has been restricted to specific types of lesions, some of these procedures have proved to be useful. However, unwarranted extension of their usage has often led to their discredit.

From the Department of Surgery of the University of Chicago, The Municipal Tuberculosis Sanatorium, Chicago, and the Stritch School of Medicine Department of Surgery of Loyola University.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

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The degree and selectivity of the collapse obtained by one of these methods; viz., extrapleural paraffin plombage, has been noteworthy. It was this observation that suggested the possible advantage of using a paraffin pack in combination with thoracoplasty.

OBJECT OF PROCEDURE

In the modern type of thoracoplasty, a serosanguineous fluid accumulates in the area of collapse following surgery. As this fluid is later absorbed, the underlying lung becomes partially re-expanded. This contributes to failure of healing of the underlying tuberculous lesion in a significant percentage of patients.

In the operation described below the space produced by the thoracoplasty is permanently obliterated by a molded piece of paraffin. This prevents re-expansion of the collapsed lung and produces a more selective type of collapse.

OPERATION

In 1947 when this procedure was first used, a two- or three-stage operation was made. Paraffin molded to fit the space produced by rib resection at the first stage was removed at the second or third stage and was not replaced. After it was found that the wax was well tolerated, it was left in place indefinitely. Furthermore, a better collapse was effected when the procedure was performed in one stage rather than in two or three stages. Since paradoxical movement was prevented by the snug fitting paraffin pack, the risk of spread of the infection was little more than in the two- or three-stage operation.

The operative procedure used at the present time (and for the last 50 cases) consists of a subperiosteal resection of the entire first and second ribs and as much of the third to the fifth, sixth or seventh ribs as indicated by the extent, location and nature of the underlying lesion. This is performed in one stage through an approach commonly employed for the conventional type of thoracoplasty. If the lesion requires more than a seven-rib thoracoplasty, the operation is performed in two stages, more paraffin being added at the second stage. Very little of the transverse processes of the vertebrae need be removed. Relaxation of the lung along the spine is increased by mobilization of the parietal pleura and division of the intercostal muscle bundles adjacent to the spine.

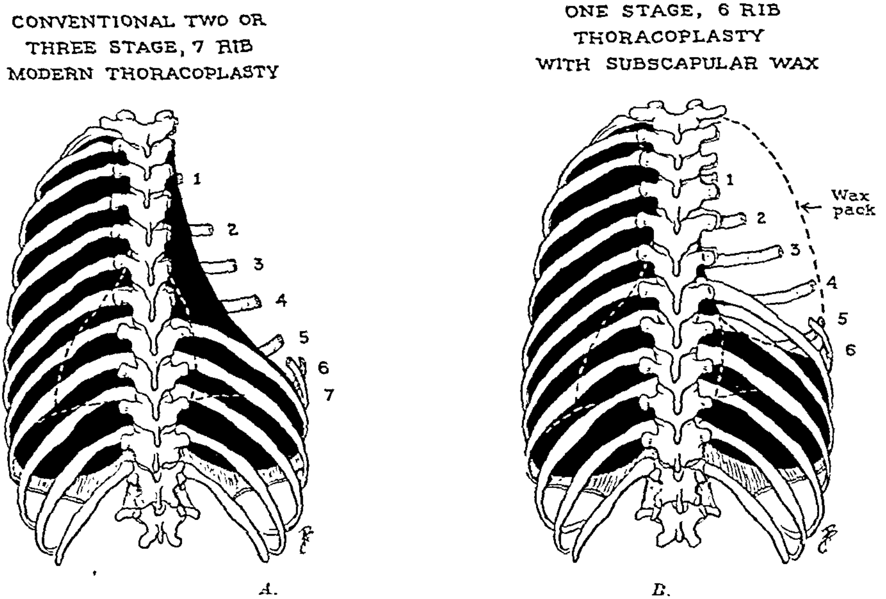
While the prepared paraffin is cooling, it is molded into one piece of a size and shape to fit the space produced by the collapse. The outer surface of the paraffin is rounded to fit into the hollow of the scapula and is flush with the outer surface of unresected ribs. No attempt is made to greatly increase the degree of the collapse by pressure.

The soft tissues of the chest wall are carefully approximated without drainage by a combination of continuous and interrupted sutures. During the immediate postoperative period a variable amount of fluid accumulates about the wax. If the amount is sufficient to cause distension of the tissues, it is removed by aspiration. This should be expected in at least 50 per cent of the operations and aspiration may need repeating.

ADVANTAGES OF THE PROCEDURE

The principal accomplishment of this operation is the production and maintenance of a more adequate collapse of a tuberculous lesion than is possible with the conventional type of thoracoplasty. This is possible due to the fact that the lung is prevented by the plomb from re-expanding as the subscapular fluid is absorbed.

Other advantages of somewhat lesser importance are: (1) a more selective type of collapse is obtained, usually associated with the resection of fewer ribs; thus, more functioning lung remains below the collapsed area, a factor of considerable importance in bilateral disease with decreased pulmonary reserve.



CONVENTIONAL TWO OR THREE STAGE, 7 RIB MODERN THORACOPLASTY

ONE STAGE, 6 RIB THORACOPLASTY WITH SUBSCAPULAR WAX

Fig. 1.—*A*, Diagrammatic illustrations of the bony thorax after a modern type, seven-rib thoracoplasty, *B*, following a six-rib, one-stage thoracoplasty with a subscapular wax pack. Note greater degree and selectivity of collapse in *B*, with resection of one less rib in one stage and without the removal of entire transverse processes.

(2) Good results may be obtained without the resection of the entire transverse processes; thus, little scoliosis develops following the procedure. (3) Since paradoxical motion is prevented by the immobile pack, a five- or seven-rib resection may be safely completed at one operation. Thus, the cost of therapy is decreased and the morbidity lowered. (4) The collapsed area is obliterated by the paraffin pack which thus prevents a sinking in of the scapula and subclavicular tissues. There is, therefore, little if any deformity following operation. Since this type of thoracoplasty has been used, there has been a noticeable willingness on the part of the patients to accept the operation.

COMPLICATIONS

Our experience has been with sixty-five patients treated over the past three and one-half years. Complications of the procedure have been infrequent and

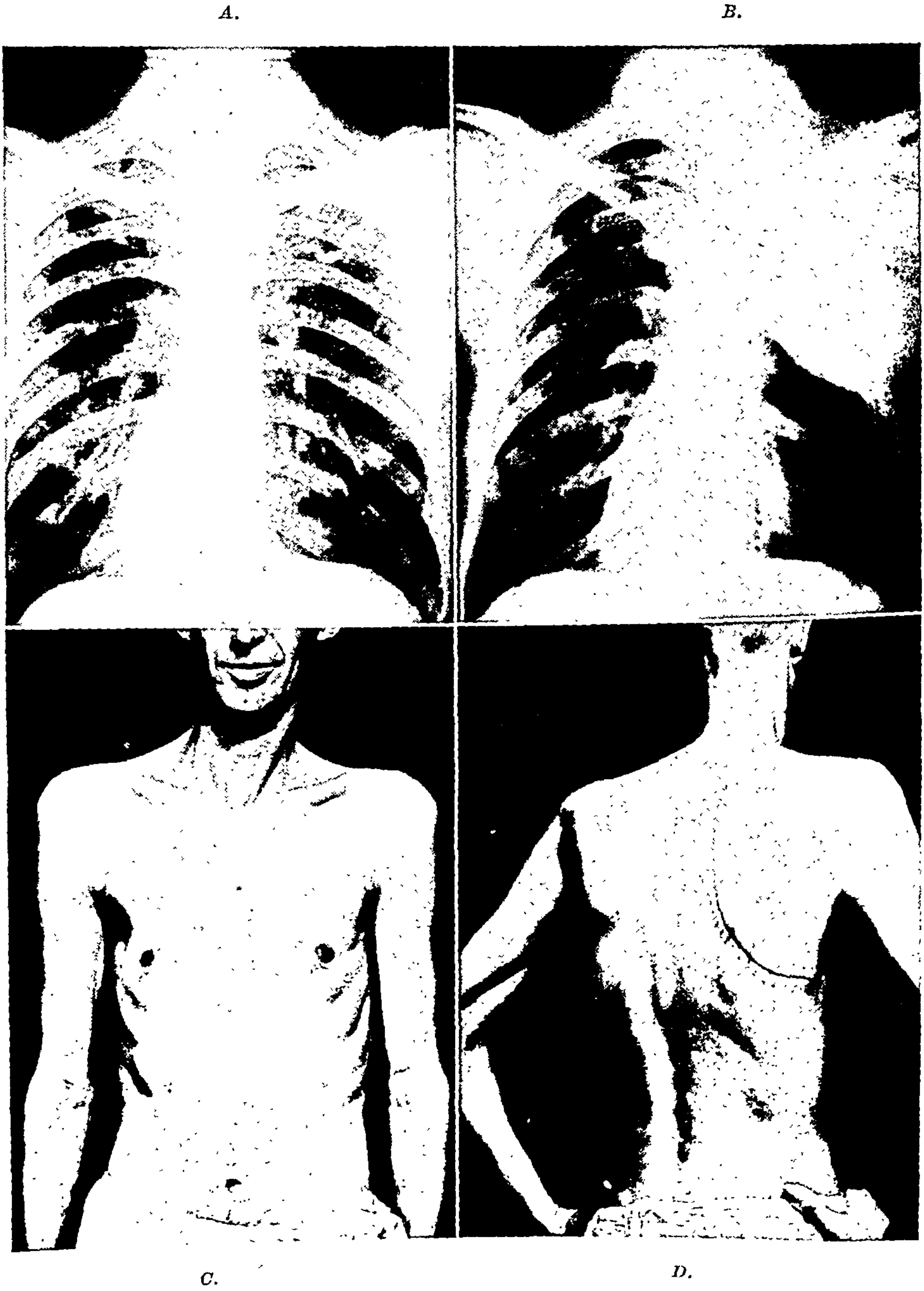


Fig. 2.—*A*, PA roentgenogram appearance of a 38-year-old white man showing multiple cavitation and infiltration extending from the apex to the level of the right second rib anteriorly. Scattered infiltrations are present in the left lung. *B*, Roentgenographic appearance following a six-rib, one-stage thoracoplasty with subscapular paraffin pack. Note selectivity and completeness of collapse with preservation of lung function below. *C* and *D*, the patient three weeks following operation. Note absence of scoliosis or other evidence of deformity.

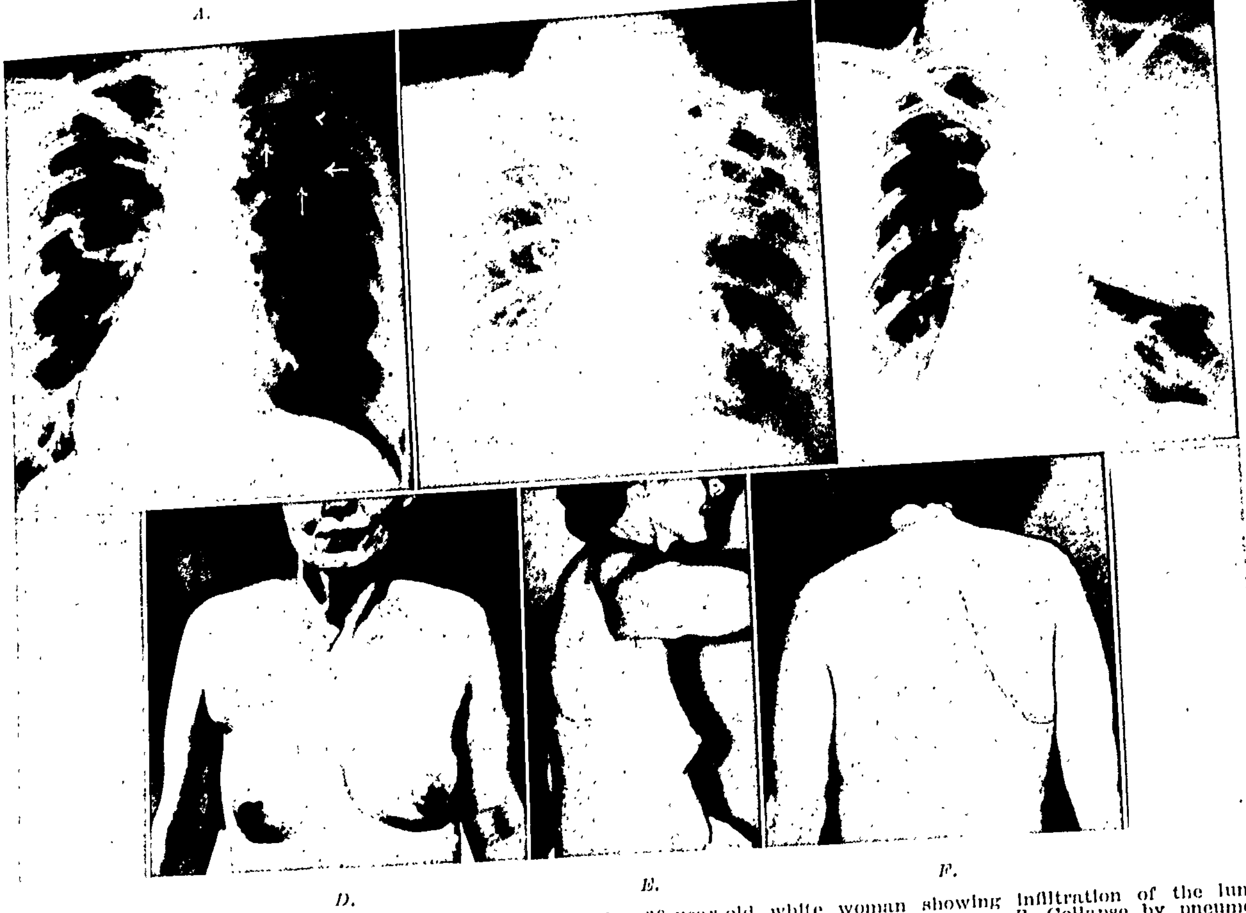
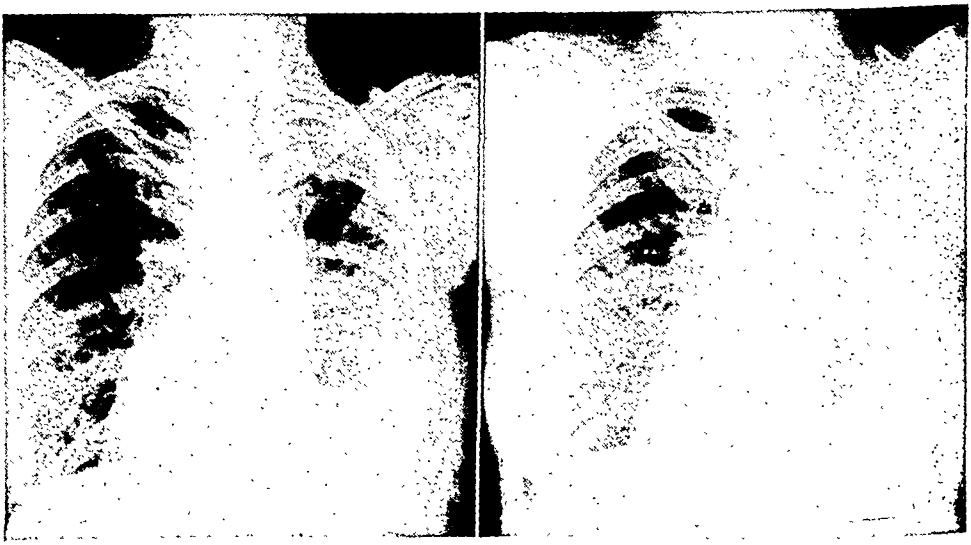


Fig. 3.—*A*, Roentgenographic appearance of chest of a 38-year-old white woman showing infiltration of the lung and two large cavities in the right apical and subapical areas. Note infiltration of left lung. *B*, Collapse by pneumothorax had been unsuccessful. *C*, Radiograph appearance following a seven-rib, one-stage thoracoplasty with subscapular paraffin pack. Note extension of selective collapse to a level somewhat below that of the resected ribs. *D*, *E*, and *F*, the patient following operation. Note little evidence of deformity.

were no different from those of the conventional type of thoracoplasty. There have been no infections of the subscapular space and no bronchial fistulas. Atelectasis has been seen in two patients, and in both instances disappeared following bronchoscopic aspiration. One superficial infection developed which healed readily without ill effect. In one patient, before aspiration of subscapular fluid was employed, opening of the wound with extrusion of a portion of the wax occurred. The wound subsequently healed spontaneously. There have been no deaths attributable to the use of subscapular wax. In 1947, one patient with far-advanced bilateral disease expired following the second stage of a two-stage thoracoplasty of cor pulmonale. In 1948 one patient died due to a transfusion reaction. There have been no late complications.



A. *B.*

Fig. 4.—*A*, Preoperative roentgenogram of a 39-year-old white woman showing moderate sized cavity in right subapical area. Incomplete aeration is present at right base following resorption of pleural effusion. Scattered infiltration is present in the left lung. *B*, Appearance following a six-rib, one-stage thoracoplasty and subscapular pack. Note completeness and selectivity of collapse.

DISCUSSION

The rationale of this operation appears to be on a sound foundation. It combines the benefits obtained by both thoracoplasty and extrapleural plombage with paraffin, and simultaneously eliminates the undesirable effects or complications of each. By protecting the lung and maintaining the blood supply to the tissues through preservation of the periosteum and intercostal structures, complications associated with extrapleural paraffin pack are avoided. Since less pulmonary reserve is sacrificed and deformity is prevented, the deficiencies of the conventional type of thoracoplasty are eliminated.

In performing the operation several factors should be kept in mind. Resection of ribs below the second should not be carried forward to include the anterior ends since this may allow the paraffin to gravitate anteriorly out of the proper position. Thus, an inferior degree of collapse would result. The amount of paraffin to be used should be of sufficient size to obliterate completely the area of collapse and maintain the scapula in normal position. This amount is usually 400 to 500 Gm. in a five- to six-rib thoracoplasty. Closure of the wound should be made with care since some pressure usually develops due to the accumulation of fluid. Closure in layers with a combination of interrupted and continuous sutures has been found satisfactory.

Indications for operation have been similar to those for modern thoracoplasty. It is possible that with further experience these indications may be extended. This is intended as a preliminary report since insufficient time has elapsed to know what may eventually be accomplished with the procedure. However, in most patients the sputum becomes negative within a few weeks after operation.

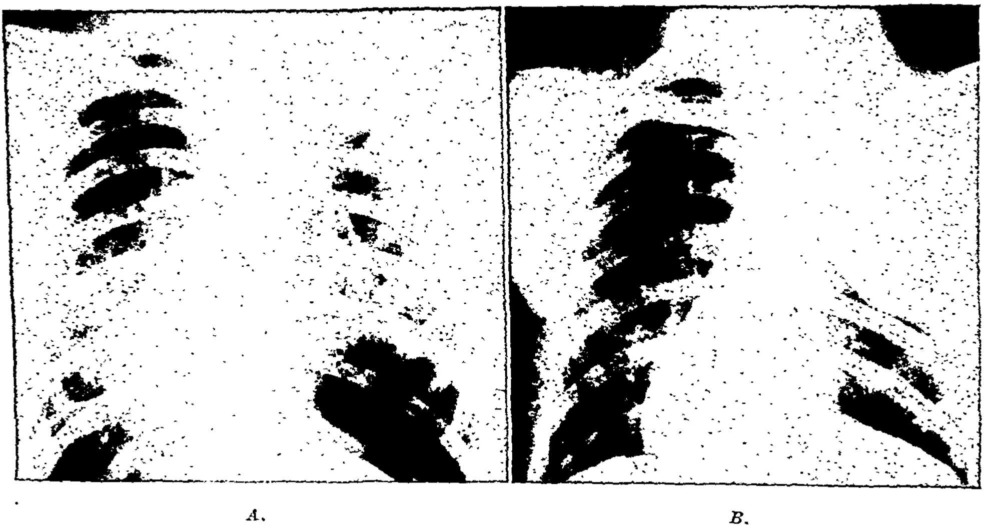


Fig. 5.—*A*, Radiograph of a 28-year-old white man showing involvement of right upper lobe with retraction toward the apex. A pneumoperitoneum is present below. *B*, Postoperative view following a six-rib, one-stage thoracoplasty with subscapular pack. The pneumoperitoneum was largely obliterated before operation.

SUMMARY

The use of subscapular paraffin pack in thoracoplasty for pulmonary tuberculosis is described from an experience with sixty-five patients over the past three and one-half years. Its advantages over the conventional type of thoracoplasty include: (1) a better and more selective collapse; (2) reduction of expense and lower morbidity; (3) less reduction in pulmonary reserve; and (4) lack of postoperative deformity. Complications are no different than those associated with the conventional type of thoracoplasty.

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DISCUSSION

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—Like Dr. Adams, we have been impressed with the possibilities of improving collapse therapy with extra-fascial packing or extra-periosteal packing. To do this, we have used plastic spheres in the extra-fascial space. The first group of patients, operated on beginning in the latter part of 1947, were those who had had concomitant removal of the upper three or four ribs.

(Slide.) This shows the reaction that occurs around the lucite spheres; it is made up largely of collagenous tissue; often the lining of this reaction is made up of low cuboidal epithelium.

(Slide.) This shows the extra-periosteal lucite pack with thoracoplasty of three ribs for closure of this cavity. This was one of the early patients who has been sputum-negative for three years.

(Slide.) Another patient, showing the cavity with the phrenic and thoracoplasty, and extrafascial lucite pack.

(Slide.) This young woman was operated on about two and a half years ago; nine months afterward she became pregnant; she was delivered without exacerbation after a normal confinement.

(Slide.) These are the results in the first fourteen cases of extrafascial lucite pack with thoracoplasty. One patient is positive, since this slide was made about eighteen months ago; two other patients have had lobectomy and one patient has expired from tuberculosis.

(Slide.) This shows the size of the cavities in which the operation was done. Most were more than two centimeters in diameter.

(Slide.) This shows the position and the technic used. Incision is almost the same as a thoracoplasty exposure.

Later we began a second type of extrafascial procedure in which we left the ribs in and put the lucite pack under the ribs.

(Slide.) This shows the lucite pack under the ribs.

Of the total number of patients who have had this type of operation, either with the ribs in or removed, there were 89, and of this number 69 or 78 per cent are sputum-negative on repeated culture at this time.

DR. EDWARD J. O'BRIEN, Detroit, Mich.—I really do not think there is much excuse for packs of any sort or embellishments of thoracoplasty. There is only one excuse for a paraffin pack, and that is because respiratory reserve is so low that nothing else could be tolerated. Here, a very small pack might be risked with proper fear of what might happen. Using these foreign bodies, whether lucite balls or snowballs or paraffin or whatever it is, reminds me of loaning money to a good friend. You expect to get it back. You will get it back either through the bronchus or when you go back in to take it out. I still think that, in a cavernous lesion, a good thoracoplasty is preferable to resection for reasons that are obvious. I think—lower mortality, fewer complications, etc. By good thoracoplasty, I mean complete resection of all the transverse processes, including first, also the rib ends. The first three ribs should be removed to the cartilage; the remaining ones far forward, and a sufficient number of them removed to get well below the cavity. If, after a good clean thoracoplasty, without a lot of rubbish piled in, you still have positive sputum after about six months or more, a resection should be done. I think the tendency is to rush to resection too quickly, however, I do not think you can expect to take a thick leather-wall cavity, filled with exudate, and collapse it, and expect it to be closed and healed in a month or six weeks. I have seen cavities that were apparently open three or four months after thoracoplasty that would close later. I think some time limit must be set, and we have thrown six months in as the probable opportune time.

I would like Dr. Adams to explain to me just how he avoids pressure when one does not know just how much of a foreign body is needed to cause cavity closure. In one of his cases, I noticed that the lesions were productive. We believe that productive, caseous lesions that persist or recur after treatment should be resected, but the use of all forms of foreign body packs and embellishments of thoracoplasty should cease.

MR. W. P. CLELAND, London, England.—I would like to thank you very much for the opportunity of speaking before you, although I rather hesitate to take the floor after Dr. O'Brien's remarks about these procedures. On the other hand, I have come to the conclusion that my "friends" must be good ones, as most of my money has not been retruned to me either through the bronchus or by other routes.

My training in the treatment of tuberculosis evolved around the Semb type of thoracoplasty, and my own experiences with that procedure have been very satisfactory, with a cavity closure rate of approximately 80 per cent and a similar rate of sputum conversion. I was, however, a little unhappy with the procedure as it necessitated multiple stages, produced a certain amount of deformity and also left the apical portion of the lung unsupported and liable to develop paradoxical movement. I believe that paradoxical movement has a lot to do with the spread of the disease and atelectasis which occurs in a number of cases.

In order to overcome some of those deficiencies I started to experiment with various forms of plombage. I read with great interest the reports from this country about the early use of lucite balls but found them rather discouraging. I think a close study of thoracoplasties and extrapleurals gives a clue to the bad results obtained with the early plombage cases. Our own experience with extrapleural pneumothoraces indicates that sooner or later about 20 per cent of cases develop tuberculous pus in that extrapleural space. On the other hand the thoracoplasty rarely develops pus in the extrafascial space. This space is more remote from the lung and therefore from diseased lung tissue and this, I think, is a clue to the avoidance of many of the troubles that one has with plombage.

I employ a procedure very similar to the one that Dr. Goldman described, leaving all the ribs in situ but stripping them of their periosteum except for a small portion on the outer aspect. This allows the intercostal muscles with their blood supply to fall down with the mobilized lung, where they form a barrier between the plomb and the lung tissue. I divide the second and third ribs posteriorly so as to give plenty of space to do an apicolysis, which I believe is of value in achieving cavity closure. After mobilizing the lung the ribs are resutured, leaving solid chest wall which does not exhibit paradoxical movement. The plastic material we have been using recently is polythene made in 1.25 inch spheres; they are solid but they have a half-inch hole bored through the center. They are in fact really beads. They are not particularly heavy but they have this great advantage over the hollow lucite balls in that they can be boiled and one can be absolutely certain about their sterility. I have done in the last few years nearly 200 cases of this type, with complication rates which are approximately half of those encountered with my own thoracoplasties; and with results as regards cavity closure and sputum conversion which I think compare favorably with the Semb type of thoracoplasty.

The whole crux of this plombage problem I am sure is going to rest upon the final attitude of the tissues to a foreign body, and in that connection I would like to mention that in these 200 cases I have had only three that have developed tuberculous pus late after the procedure. These are the only three cases that have given rise to any trouble directly referable to the plomb.

DR. T. R. HUDSON, Chicago, Ill.—Being situated geographically rather close to Dr. Adams, Dr. Head and I were stimulated early in his work and we have tried this procedure; altogether we have used it in forty cases. We have followed a somewhat different procedure, however, in that we ordinarily do not leave the paraffin permanently in place, removing it at the second stage of thoracoplasty. For one reason or another, however, in fifteen cases we did leave paraffin in place after the first stage of thoracoplasty. Four of these had to be removed because of unusual accumulations of fluid which could not be effectively dealt with by repeated thoracentesis. So far as complications are concerned we have had none in these forty cases, unless you consider a rather violent accumulation of fluid around the pack a complication. There have been no infections, no extrusion of paraffin, and no bronchopleural fistulae. I would like to show three or four slides:

(Slide.) This series of slides simply shows the lesion at the beginning.

(Slide.) This shows the paraffin pack in place, and the third slide shows the patient after the second stage of thoracoplasty and removal of the paraffin pack.

I think you will all agree that is a rather severe lesion shown in the *first film*, and probably a much better thoracoplasty with the pack than one would expect without having used it.

(Slide.) This is another patient; on the left the appearance of the chest after the second stage of thoracoplasty, the pack having been removed; it shows the large accumulation of fluid that does occur.

(Slide.) This shows the girl after an undetermined period, and shows, I believe, a rather good thoracoplasty and one which might be better than one would expect without the use of the pack.

(Slide.) This shows a girl with a very large cavity in the top of the left lung, in which we did two stages of thoracoplasty, using the pack between the stages. This lesion was so extensive that we felt something in addition was needed, so, following removal of the pack in the second stage we continued to keep this space by means of air injections into the subscapular space, and the second slide shows her condition during that course of treatment.

I might say that in removing all these packs we have noticed a very definite inflammatory membrane that forms around the pack, and I suppose it is this inflammatory membrane that contains the air and keeps it from extravasating into the fascial and muscular planes.

(Slide.) This is an example of a patient in whom we had thought we might leave the pack in, but as you see there was a terrific extravasation of fluid around the pack; the operation was done originally for a small lesion, and it was felt unjustified to leave the lung in this condition, so we removed the pack and continued the smaller refill space which does effectively control the lesion.

DR. ROBERT KLOPSTOCK, Jamaica, New York.—It is recognized that the most reliable form of cavity healing occurs by concentric shrinkage and not by compression and subsequent inspissation. We felt that the mechanism for this method of cavity closure could be achieved by providing a graded relaxation of the chest wall rather than by added compression of the diseased area. Performing the thoracoplasty in caudo-apical progression seems to provide the mechanism referred to.

The advantage of this procedure was brought to our attention by Drs. Herbert C. Maier and Walter Fischer who, in 1947, demonstrated a case with a giant cavity in which the thoracoplasty was performed from below upward to prevent ipsilateral spread. We were so impressed with their result "as such" that we undertook caudo-apical thoracoplasty in a number of consecutive cases and studied the mechanism of cavity closure with interstage anteroposterior and lateral tomography. We found that the so-called lower or first stage (at a level as determined by the extent of the pathology) provides an immediate relaxation of the diseased area and an immediate concentric diminution in the size of the cavity. And at the time of the final or apical stage, it permits a concentric shrinkage of the cavity-bearing parenchyma rather than causing compression of the cavity itself and its subsequent inspissation. Our observation is based on 100 consecutive cases performed at Triboro and Seaton Hospitals by Morris Rubin and myself, on patients in whom resection was not desirable due to their borderline conditions or to the extent and distribution of their pathology. This procedure, as we used it, eliminated the need for supplementary procedures such as apicolysis, anterior stages, or revisions. The patients have been followed from one to four years since completion of their surgery.

(Slide.) These are roentgenograms of a typical case operated upon; the preoperative conventional chest radiogram, and left lateral tomogram. You see two very large cavities; one occupying the apico-posterior segment of the left upper lobe, one in the superior segment of the left lower lobe.

(Slide.) The Bucky film of the left chest shows that the posterolateral segments of the seventh and sixth ribs have been resected. You see on the two lateral tomograms that both cavities are markedly diminished in size by concentric shrinkage.

(Slide.) Again the Bucky film of the left chest: lower and middle stages have been completed and the upper three ribs not yet removed. The two lateral tomograms demonstrate a further considerable decrease in the size of the cavities.

(Slide.) This is the conventional roentgenogram of the chest after completion of the seven-rib thoracoplasty. The patient has been observed now for three years, having negative cultures.

We feel that such gratifying results in the presence of large cavities of the size as demonstrated could not have been achieved with a seven-rib thoracoplasty if developed in the conventional apico-caudal fashion.

DR. ADAMS, Chicago (closing).—I wish to thank the discussors for their remarks. I appreciate the fact that the reaction against using foreign bodies within the chest has been rather severe, and naturally one is influenced by his experiences which may have been unfavorable in the use of foreign material. This has been largely in connection with extrapleural paraffin pack or lucite plombage. I was acquainted with the experiences of the group at the Brompton Hospital, having talked with Dr. Lucas a short time ago when he visited this country. Others including Dr. Goldman, Dr. Head, and Dr. Meltzer, have used paraffin or plastic material placed outside of the intercostal muscles and periosteum beneath the scapula, with favorable results. The results, as time goes on, are likely to be different from those in cases where it was used extrapleurally. Of course, as I have said, this is in the form of a preliminary report and at a later date we can make a follow-up report.

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ENDOSCOPIC SPONGE BIOPSY

M. G. CARTER, M.D.* (BY INVITATION), R. R. NESBIT, M.D.** (BY INVITATION),
AND C. A. PIPER, M.D.*** (BY INVITATION)
NEW HAVEN, CONN.

RECENT improvements in both the instruments and technique of endoscopy and the introduction of methods for cytologic examination of sputum and bronchial secretions have reduced the number of patients with intrathoracic disease in whom a preoperative diagnosis cannot be made. We have used modifications of the Papanicalaou smear technique for identification of cells secured at bronchoscopy or esophagoscopy. There are certain limitations to the method, as others have noted.[1] Our principal troubles were inadequate specimens or smears which were too heavy, excessive amounts of pus or mucus, a predominance of blood in the slides, and dry specimens. There is no question that the pathologist must be interested in spending time and effort in the search for and interpretation of cells, which at times is a tiresome and difficult task. Even under the best circumstances, the limitations of this, and probably of any other cytologic method, prevent either the dogmatic assumption that positive smears always mean cancer, or more certainly, that negative smears prove its absence.[2]

Recently, we began using Gelfoam, and later Onkosponge† for collection of secretions endoscopically. This report summarizes the significant information from the first eighty patients in whom bronchoscopy and/or esophagoscopy were performed since we adopted this cytologic method.

METHOD

Sponge biopsies were obtained endoscopically by the method described by Gladstone.[3] They are secured at bronchoscopy as illustrated in Fig. 1. The bronchoscope is passed and, after a thorough examination of the bronchial tree, a small segment of sponge which has been securely fastened to the standard sponge carrier is introduced into the orifice of the suspected lobe or segment. This sometimes induces cough, which is probably a desirable effect, since it ejects material from the bronchus. The sponge is moved gently and is allowed to remain in contact with the secretions or mucosal surface for at least one minute. It is then withdrawn and immediately placed in a small bottle containing 10 per cent neutral formalin. If a visible tumor is present, the sponge may be rubbed vigorously over the tumor surface.

When investigating lesions of the esophagus or cardiac end of the stomach, the sponge is rubbed over the visible tumor or pushed through the strictured area. If cancer is thought to penetrate the wall of the trachea secondary to an esophageal lesion, the sponge is rubbed over the area suspected.

From the Thoracic Service and the Department of Pathology, Hospital of St. Raphael.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

*Assistant Visiting Surgeon in Charge of Thoracic Surgery, Hospital of St. Raphael.

**Pathologist, Hospital of St. Raphael.

***Lieutenant, U. S. Navy Medical Corps. Formerly, Resident Surgeon, Thoracic Service, Hospital of St. Raphael.

†May be obtained from Histomed, Inc., Patterson, N. J.

When received by the pathology laboratory, the sponge is handled precisely as are tissue sections. After removal from the formalin it is carried through the fixing fluids, embedded in paraffin, and multiple sections are made which are placed serially on one or more slides. The paraffin blocks are preserved so that further sections may be cut if interpretation of the slides is equivocal.

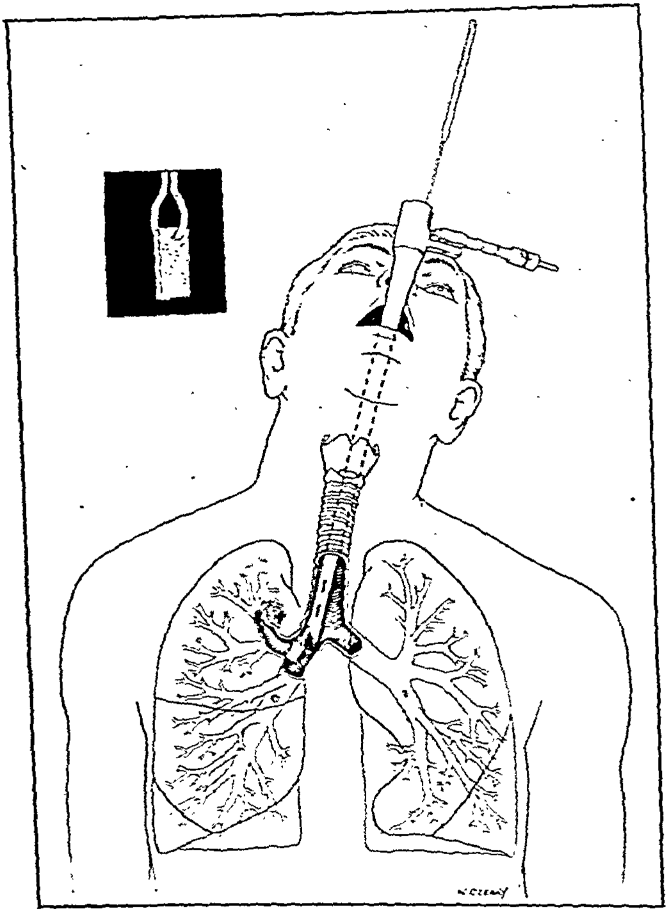

Fig. 1.—Drawing to illustrate the sponge biopsy method for obtaining tumor cells at bronchoscopy. Inset shows enlarged view of sponge in carrier.

MATERIAL AND RESULTS

The ages of the patients ranged from 20 to 81 years. Fifty-nine of the patients were over 50 years of age. There were sixty-five males and fifteen females.

Twenty-three patients were proved to have tumors of the lung of which twenty-two were cancer and one was adenoma. The diagnostic sequence for these patients is illustrated in Table I. Preliminary clinical diagnoses included carcinoma in twenty-one patients, lung abscess in one patient, and pneumonia in one patient. A positive diagnosis of cancer was established by bronchoscopy in

TABLE I. DIAGNOSTIC SEQUENCE IN TWENTY-THREE CASES OF PROVED PULMONARY TUMORS

	DIAGNOSES					
DIAGNOSTIC METHODS	ABSCESS	PNEUMONIA	CANCER	SUSPECTED CANCER	BENIGN TUMOR	NO TUMOR SEEN
Clinical	1	1	21			
Endoscopic	1		7	7		8
Sponge			7	6	1	9
Final diagnoses*			22		1	

*Established by operation or autopsy.

seven, suspected in eight, and bronchoscopy was negative in eight. In this same group, sponge biopsy was positive for cancer cells in seven and questionable in six. No tumor cells were seen in nine, and tumor cells "probably benign" were found in one. This last patient was subsequently shown to have a bronchial adenoma. The patients with a positive sponge biopsy were not all from the same group of seven as those showing a positive bronchoscopy. Tumor cells were identified in the sponge taken from two patients in whom tumors were not diagnosed by bronchoscopy. There were no falsely positive sponge biopsies.

TABLE II. DIAGNOSTIC SEQUENCE IN FOURTEEN PATIENTS WITH LESIONS OF THE ESOPHAGUS OR GASTRIC CARDIA

	CANCER OF ESOPHAGUS	CANCER INVADING TRACHEA	CANCER GASTRIC CARDIA ASCENDING	ESOPHAGEAL STRICTURE, BENIGN
Endoscopy diagnostic	9	1 (Fistula)	0	2(?)
Endoscopy not diagnostic	0	2	3	0
Sponge diagnostic	Not done	2	1 (Suggestive)	1 (Suggestive)
Total cases	9		3	2

Fourteen patients had lesions in the esophagus or cardiac end of the stomach (Table II). Nine of the esophageal lesions proved to be carcinoma, and in three of these the tumor had eroded into the trachea. Invasion of the trachea could not be confirmed with certainty by the bronchoscope alone, but sponge biopsies from the trachea of two patients revealed cancer cells. In one of the three patients with tracheal invasion a fistula was present and the sponge was unsatisfactory. The diagnosis of benign stricture of the lower end of the esophagus was made in three patients who later proved to have carcinoma of the gastric cardia. In one of these, cells suggesting carcinoma were present on the sponge; the sponge was negative in the other two.

Two patients with benign stricture of the lower one-third of the esophagus, secondary to peptic esophagitis, were also examined. In one of these, hyperkeratosis of the cells was noted.

Twenty-two patients had acute and chronic pulmonary inflammatory diseases. These included bronchiectasis, bronchitis, middle lobe syndrome, infarct, pneumonia, and lung abscess. In this entire group there was not a single sponge biopsy which was interpreted to show tumor cells (Table III).

Three patients had pulmonary tuberculosis. In two, this was parenchymal and in one there was tuberculous endobronchial ulceration. Endoscopic sponge biopsies showed pus cells, bacteria, and fungi, but no tumor cells. Acid-fast stains

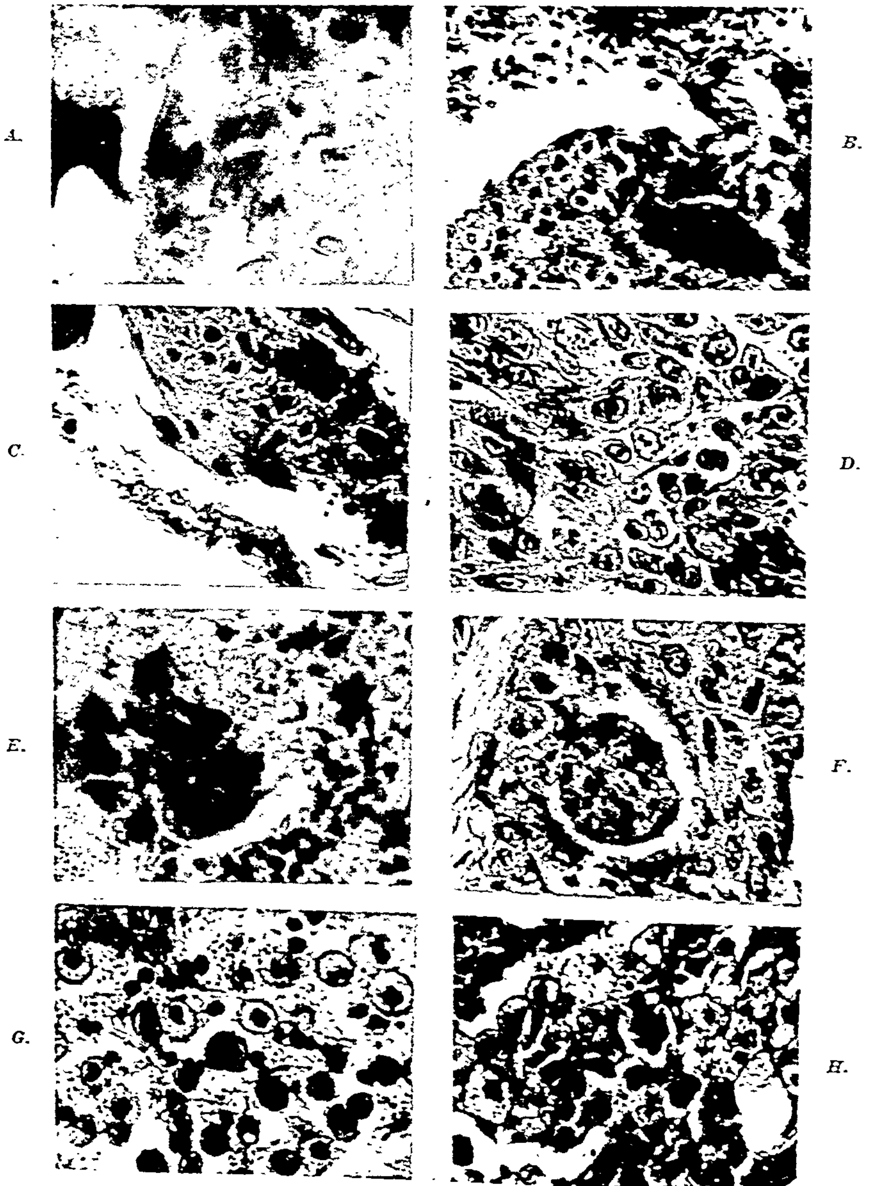

Fig. 2—*A*, Gelfoam sponge with hyperkeratotic epithelial cell nest from a case of lung abscess (middle lobe syndrome). *B*, Tissue section from the same case, showing bronchial metaplasia. *C*, Nest of epidermoid carcinoma cells in sponge taken from trachea at site of invasion by carcinoma of esophagus. *D*, Biopsy of esophagus from the same case. *E*, Malignant cells in sponge biopsy from patient with metastatic tumor eroding bronchus. Note large numbers of red cells surrounding tumor nest. *F*, Section from excised lung showing tumor (primary site, corpus uteri). *G*, Bronchial sponge biopsy showing varying phases of adenoma cells (compare to cells in *H*). *H*, Section from lung showing development of similar cell forms within tumor.

TABLE III. INFLAMMATORY DISEASES IN WHICH SPONGE BIOPSY WAS NEGATIVE (25 CASES)

Bronchiectasis	7
Bronchitis	4
Middle lobe syndrome	3
Infarct	1
Pneumonia	4
Lung abscess	3
Tuberculosis	3

done on these sections did not reveal tubercle bacilli even though the sputum was heavily positive in every case. The remaining 18 patients encompassed such diseases as atelectasis, emphysema, silicosis, cardiospasm, myxedema, and psychogenic disorders. In none of these was a falsely positive sponge biopsy obtained.

DISCUSSION

We have been impressed with the ease of collection, preparation, and the microscopic excellence of material prepared by the sponge method. Fig. 5 illustrates sponge biopsies compared with sections obtained either by tissue biopsy or surgical extirpation. It is apparent that the cells to be examined are confined to a limited area of the slide, and sections at multiple levels can be made to investigate suspicious areas. There is uniform depth of sections, and the difficulty of overlapping pus and blood cells is thereby minimized. The standard tissue stain with which the pathologist is familiar can be used for these sections, and the interpretation therefore requires no familiarity with special stains.

The number of tumors in this series is too small for statistical significance. We were impressed, however, with the absence of falsely positive slides as well as by the positive cytologic diagnoses in two patients with negative findings at bronchoscopy. In these two cases the secretions sopped up by the sponges were the only means of making a correct preoperative diagnosis. Earlier in the series we compared the sponge method with the Papanicalaou method in each case, but shortly abandoned the latter because superior slides were obtained by the sponge method.

A sponge biopsy is usually not necessary for the diagnosis of esophageal carcinoma, since the tumor is almost always visible and a direct biopsy may be taken. Lesions of the gastric cardia, however, often extend submucosally upward into the esophagus without presenting a mucosal tumor. In these cases the sponge passed through the strictured area may pick up tumor cells. This was accomplished in one of our cases. Of perhaps more importance is the demonstration of direct tumor invasion in the posterior membranous portion of the trachea from a primary site in the esophagus. In two of our patients in whom this was suspected, no endotracheal tumor was visible but the sponges taken from the trachea contained tumor cells. Inoperability was therefore established without recourse to thoracotomy.

The differentiation between pulmonary suppurative disease alone and such inflammatory disease secondary to the growth of tumors is of vital importance. We were pleased that in our twenty-two patients with inflammatory diseases of the lungs there were no falsely positive sponge biopsies. Accurate diagnosis in this type of patient is difficult either by sputum or endoscopic Papanicalaou examination because of the large numbers of inflammatory cells, and frequently red cells as well. The paraffin block method of preparation allows one to discriminate more carefully between inflammatory cells and those cells desquamated from tumors.

We have learned several precautions which must be observed in order to obtain satisfactory sponge biopsies. First, the sponge must remain in contact with the secretions for a sufficiently long time to allow absorption of a large number of cells. The minimum time appears to be approximately one minute. Second, when the nurse is sectioning the sponge and placing it on the sponge carrier, great care must be used not to compress the sponge with the fingers and therefore to destroy its absorptive qualities. Finally, of course, the sponge should be placed immediately in fixing solution so that there will be no distortion of the cells by drying. Greatest diagnostic accuracy may be expected when the sponge is rubbed directly on the tumor. This is not often possible endoscopically. When it is, one should also be able to obtain a direct biopsy.

We feel that the comment of Ulfelder applies to this method as well as to that of Papanicalaou, "The results are most accurate when slides are prepared by a person interested in the procedure and willing to give time and attention to details that may facilitate interpretation, and when the slides are examined by someone thoroughly familiar with the cellular pattern of that particular fluid, prepared in that particular way."[4] Other cytologic methods may be most satisfactory when handled by other people, but in our hands, sponge biopsy has proved of greatest aid.

SUMMARY

The sponge biopsy technique has been applied in a series of eighty consecutive patients subjected to diagnostic bronchoscopy or esophagoscopy. It was found useful particularly for the identification of pulmonary tumors, the distinction between inflammatory disease and tumor, the delineation of esophageal cancer which had extended into the trachea, and on one occasion for the identification of tumor cells from a gastric cancer with submucosal esophageal extension. In our experience, the technique is simple and produces microscopically more readable preparations than other currently used endoscopic cytologic methods of diagnosis. The technique of sponge biopsy is described and illustrative photomicrographs are presented.

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THE PATHOGENESIS OF BRONCHOPLEURAL FISTULAS FOLLOWING EXCISIONAL THERAPY FOR PULMONARY TUBERCULOSIS

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BRONCHIAL stump fistula accompanied by empyema is one of the most common serious complications of excisional therapy for tuberculosis. The infection often resembles the mixed infection tuberculous empyema which follows the formation of pulmonary bronchopleural fistulas. Such a fistula is a serious threat to the life of the patient, since aspirated material from the infected chest space may be the source of an overwhelming spread of the disease to the remaining lung parenchyma. It is the purpose of this paper to describe the mechanisms by which this condition may develop.

MATERIAL

We have encountered nine bronchopleural fistulas in 84 (10.7 per cent) postoperative tuberculous patients who had been treated by excisional therapy. The clinical and pathologic findings in five of these cases are described in detail, since we believe that their fistulas arose in a manner which has been subject to some controversy. Four of these five cases were studied at necropsy and one is alive and well; three of the patients had pneumonectomy, one had lobectomy, and one had a segmental resection. Since the cause of the fistulas in two cases is not subject to question, they are included in the body of the discussion which follows the case reports. Both patients had segmental resections and are still alive. The other two patients died, but are not included since they were not studied at necropsy.

CASE REPORTS

CASE 1.—This 20-year-old Negro man was discovered to have pulmonary tuberculosis in 1947. He had been well until June, 1947, when he developed malaise, fatigue, weight loss, and cough. Roentgenogram on June 26, 1947, revealed a mottled area involving the entire left upper lung field. All sputa were positive for acid-fast bacilli. Streptomycin was begun on Aug. 5, 1947, at a dosage of 0.4 Gm. every three hours. This was changed to 0.5 Gm. four times a day on Aug. 5, 1947, and continued until Nov. 5, 1947, at which time he had received a total of 200 Gm. He became afebrile and began to gain weight. A left phrenic crush was performed on Aug. 1, 1947. On Aug. 13, 1947, a left pneumothorax was induced but was ineffective because of pleural adhesions. On Jan. 28, 1948, he developed scrotal pain. This was accompanied by swelling of the left epididymis. A diagnosis of tuberculous epididymitis was made and the patient was given 38 Gm. of streptomycin in

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From the Laboratory and Surgical Services, Halloran Veterans Administration Hospital.

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thirty-eight days. His symptoms subsided and the swelling abated. He was admitted to the Halloran VA Hospital on April 20, 1948, at which time his sputum was Gaffky V. The organisms were sensitive to 1 μg of streptomycin. Roentgenograms revealed an area of rarefaction above the left second anterior rib.

In June, 1948, he again became febrile, and roentgenogram revealed a spread to the left lower lobe. In view of failure of previous collapse therapy and the ineffective cough due to a permanent left phrenic crush, it was felt that resection was the treatment of choice. The right lung appeared stable. He was started on a third course of streptomycin on July 23, 1948, at a dose of 0.5 Gm. twice a day.

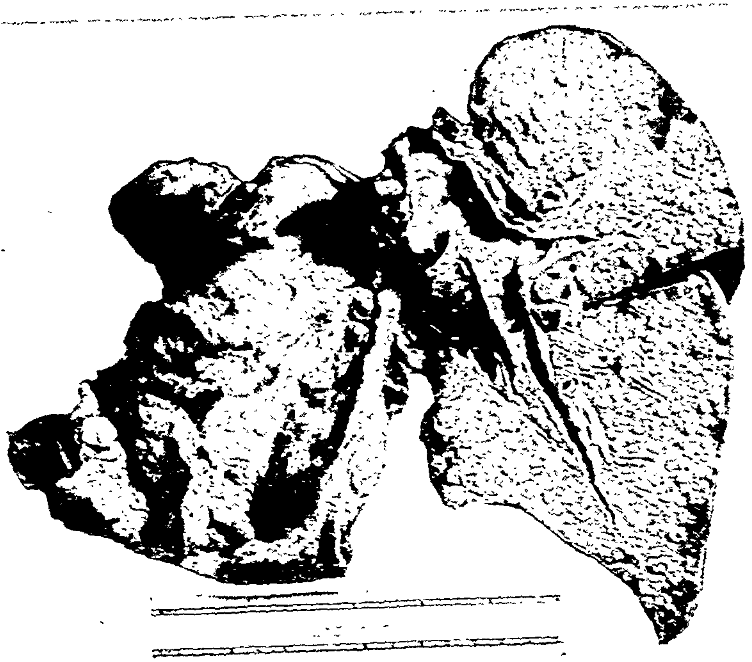

Fig. 1.—(Case 1) Tuberculous empyema with stump fistula. Note the 1 cm. segment of bronchial stump, the opening at the distal end of the stump and the large numbers of fresh tuberculous lesions in the intact lung.

A left pneumonectomy was performed on Aug. 24, 1948. It was performed with moderate difficulty and early control of the bronchus. The pleural space was obliterated by dense adhesions. The lung was contracted and fibrotic. After removal of the lung, traction sutures were placed proximal to the clamped stump which was then reamputated with interrupted sutures of 000 silk. The stump was buried with a pleural flap. The wound was irrigated with saline and the chest was closed without drainage after the instillation of 200,000 units of penicillin and 1 Gm. of streptomycin.

The patient appeared to be doing well for the first two postoperative days. On the third day he spiked a fever of 102° F. This fever continued until the twenty-first postoperative day. During this period it was necessary to aspirate an unusual amount of fluid from the left chest (3090 c.c. of serosanguineous fluid from Aug. 28, 1948, to Sept. 16, 1948, and 755 c.c. from Sept. 17, 1948, to Oct. 5, 1948). Culture of this fluid on Sept. 30, 1948, was positive for *M. tuberculosis*. On Oct. 4, 1948, *Staph. aureus hemolyticus* was also cultured from the fluid. Streptomycin and penicillin were instilled into the pleural space every other day, and subcutaneous injections of streptomycin were also continued. On the forty-first postoperative day the patient spiked a fever of 103° F., and coughed up reddish-brown material, establishing a diagnosis of bronchopleural fistula. Attempts to control the fistula with intercostal underwater drainage were unsuccessful. The patient now developed a massive spread to the right upper lobe. The chest was reopened, and a 4 mm. stump fistula was found. This was closed with a pectoralis major muscle flap. The fistula remained closed for only three days. Respiratory insufficiency developed, and the patient died on Oct. 30, 1948 (sixty-seventh postoperative day).

Anatomic Findings.—

A. *Surgical Specimens:* Left Lung: Tuberculous cavities were present in both lobes of the left lung. Encapsulated caseous foci, which were softened centrally, were present in the lower lobe. Partially healed acinous nodose foci were present in both lobes. Although the pleura overlying the lung was thickened, there was no evidence of pleural tuberculosis. The left main stem bronchus was free of disease at its cut edge.

B. *Necropsy Findings:* Gross Examination: The left pleural space contained 200 c.c. of creamy yellow pus. The parietal pleura (Fig. 1) varied from 2 mm. to 1.5 cm. in thickness. It was lined by a layer of shaggy material which varied from yellow to red-brown in color. A 3.5 cm. sinus tract led from the pleural space to a bronchial stump measuring 1 cm. in length. The lining of this tract was similar to that which lined the pleura. The muscle flap hung free within the pleural space. The mucosa of the lower trachea and both main bronchi had a red granular appearance and contained many depressions from 1 to 4 mm. in diameter. These were most numerous in the stump of the left main bronchus.

The tracheobronchial lymph nodes adjacent to the stump were mottled gray-white and slate gray-black in color. They contained no caseous foci.

Scattered throughout the right lung were many acinous nodose foci, and areas of caseous lobular pneumonia which varied from 5 mm. to 3 cm. in diameter. These areas were most numerous in the medial and basal aspects of the lung. There were no cavities or encapsulated foci in any of the lobes of the right lung.

The mucosa of the larynx, and of the small intestine and of the colon, was intact on gross examination. The left testis was occupied by dense gray tissue.

Histologic Examination: The left pleural space was lined by a pyogenic membrane. Beyond this was a wide granulation tissue containing epithelioid cells and Langhans giant cells. Deep to this was dense collagenous connective tissue. Contained within the granulation tissue and the connective tissue in deeper portions of the pleura were large and small tuberculous foci. The former had caseous centers. Deep tuberculous ulcers were present at the carina of the trachea and within the left main bronchus. The adventitial aspects of these structures were markedly thickened, and contained many round foci. The largest of these had a central zone of caseation. Similar foci were also present in the adventitial aspects of the adjacent esophagus.

There were few healing changes about the acinous nodose foci and areas of caseous lobular pneumonia. Both types of foci were composed for the most part of caseous material containing many nuclear fragments. They were surrounded by a wide perifocal reaction.

Summary.—This 20-year-old Negro man developed a tuberculous empyema after a left pneumonectomy. On the forty-first postoperative day he developed a bronchopleural fistula. This was followed by rapid spread to the contralateral lung. At necropsy the oldest tuberculous lesion was present in the left pleural space. The right lung was free of all but recent lesions derived by bronchogenic spread from the left pleural space. As in subsequent cases the age of the lesions was estimated on the basis of the amount of healing change in the adjacent tissue.

CASE 2.—This 26-year-old white man was found to have tuberculosis in August, 1946, when it was noted that he had a cavity in the right upper lobe. He was treated at another hospital by an eight-rib thoracoplasty. This was accompanied by a ninety-eight day course of streptomycin at a dosage of 0.3 Gm. twice a day for a total dose of 58.8 Gm. The collapse obtained by thoracoplasty was poor. The regenerating ribs assumed a convex outline, and marked scoliosis developed. The cavity remained open and the sputum remained positive. The patient left the hospital against medical advice. Sputa examinations done while the patient was at home were said to be negative. He entered Halloran VA Hospital in October, 1949, after sputum examination revealed the presence of acid-fast bacilli. Roentgenograms revealed two cavities in the right infraclavicular region associated with considerable exudative disease. The left lung appeared free of disease.

Because of the failure of collapse therapy a right pneumonectomy was performed on Jan. 13, 1950. This was done without undue difficulty. The volume of the right pleural cavity was reduced one-third by the previous thoracoplasty. There was pleural symphysis at the apex and at the superior segment of the lower lobe. There was a prolonged extrapleural dissection prior to securing the bronchus. It was not felt that considerable spill occurred when the bronchus was divided. The vessels were then isolated, ligated, and divided. The lung was then removed. The bronchus was freed of the peribronchial tissue. Traction sutures of 00 were placed posterolaterally and posteromedially. The stump was amputated at a higher level and closed with interrupted 000 cotton sutures.

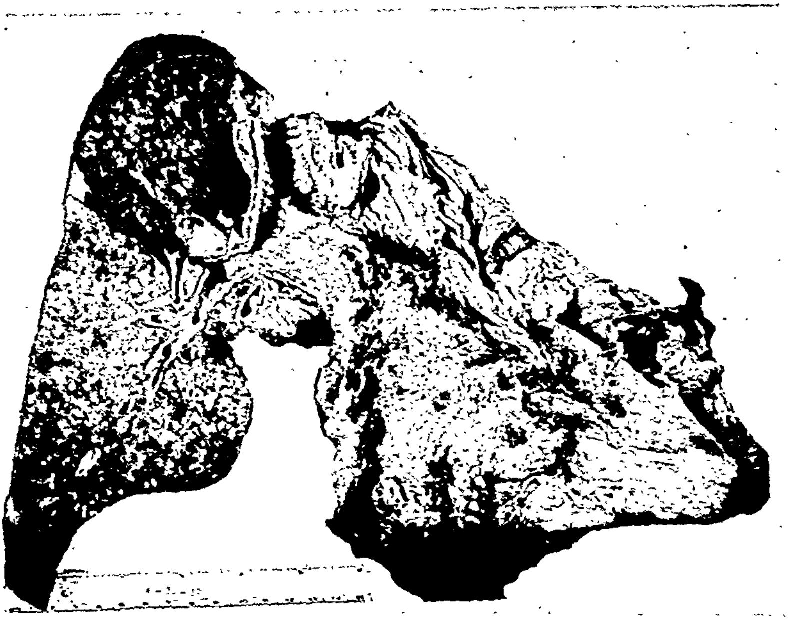

Fig. 2.—(Case 2) Tuberculous empyema with stump fistula. Note the 1 cm. segment of bronchial stump, the opening at the distal end of the stump and the large numbers of fresh tuberculous lesions in the intact lung.

Gelfoam was placed in apposition to the stump which was then buried by apposition of the mediastinal pleura to the periesophageal tissue. The pleural cavity was washed with 2,000 c.c. of saline and 30 c.c. of saline containing 1 Gm. of streptomycin and 200,000 units of penicillin. A clamped intercostal catheter was left in place inferiorly and posterolaterally. It was felt that he stood the operation well. Postoperatively he ran a fever which varied from 100° F. to 102° F. for two weeks. Culture of chest fluid at this time failed to grow *M. tuberculosis* or pyogens. The patient remained afebrile from Jan. 28, 1950, to Feb. 5, 1950 (twenty-third postoperative day). He then began to run an evening fever which rose to 100.4° F. A bronchopleural fistula was demonstrated on the twenty-eighth postoperative day. Review of chest films up to this date revealed that the left lung remained free of disease. Streptomycin was given in a dose of 0.5 Gm. twice a day, commencing on Feb. 10, 1950. The patient was bronchoscoped on February 10, and a 4 mm. opening was observed in the bronchial stump. This was cauterized with 25 per cent silver nitrate. This was repeated on February 16 and February 23, but did not stimulate bronchial closure. On February 23 the tenth rib was excised and the pleural space was packed with gauze soaked in streptomycin and penicillin. Roentgenogram at this time revealed massive spread to the left lung. He went into respiratory failure and succumbed on Mar. 11, 1950 (fifty-seventh postoperative day).

Anatomic Findings.—

A. *Resected Right Lung:* Gross Examination: Two slitlike cavities were present in the posterolateral aspects of the upper lobe. These were lined by yellow cheesy material. Upper lobe bronchi opened widely into both cavities. The cavity wall varied from 2 to 4 mm. in thickness. The intervening parenchyma was traversed by bands of gray tissue which gave the lung in this region a solid consistency. Scattered through all lobes of the lung were many round encapsulated foci from 2 to 4 mm. in diameter. Acinous foci were present in the lower lobe. There was a pleural symphysis over the apex of the upper lobe and over the posterior, superior, and lateral aspects of the lower lobe. The combined pleurae measured 2 mm. in thickness in these regions, and were composed of dense gray tissue. The peribronchial lymph nodes varied from 5 mm. to 1.4 mm. in diameter, and on section were slate gray-black. The right main bronchus showed no evidence of gross pathologic change.

Histologic Changes: there was no microscopic evidence of tuberculosis in either the pleura or the right main bronchus. One small tuberculous focus was found in a peribronchial lymph node. The cavities were lined by a wide zone of caseation. Small round foci were present within the submucosa of the bronchi draining these cavities.

B. *Necropsy Findings:* Gross Examination: The right chest cavity was lined in part by friable yellow material and in part by a red granular tissue (Fig. 2). The cavity wall was composed of firm tissue which measured from 2 to 5 mm. in thickness. This tissue blended with the muscles of the chest wall, with the diaphragm, and with the tissue of the mediastinum. There were many small round yellow foci within the mediastinal soft tissue adjacent to the pleura. A 1.1 cm. segment of right stem bronchus remained intact. A 5 mm. opening at its distal end communicated with the pleural space. The mucosa of both stem bronchi had a granular appearance. Scattered throughout both lobes of the left lung, but most numerous in the lower and medial aspects, were many acinous nodose foci. These measured from 1 to 7 mm. in diameter. The tracheobronchial lymph nodes varied from 5 mm. to 4 cm. in diameter, and on section were slate gray-black. Numerous small round foci surrounded the right inferior tracheobronchial lymph node.

Histologic Appearance: the pleural space was lined by a pyogenic membrane. Deep to this was an edematous vascular granulation tissue containing epithelioid cells and Langhans giant cells. Beyond this was a dense collagenous connective tissue. Numerous small round foci were present in the granulation tissue and within the dense connective tissue. The epithelium of the bronchial stump was flattened where it joined the pleural space. Many small foci were present deep within the bronchial wall at this point. Collections of caseous material were present in the lumina of the bronchioles of the left lung. The walls of some of these bronchioles and portions of the adjacent lung also were caseous. These small focal areas were surrounded by a scanty vascular granulation tissue composed of epithelioid cells, Langhans giant cells, fibroblasts, polymorphonuclears and collagen fibers. Occasional small round submucosal tuberculous foci were present in the left main-stem bronchus and within the tracheobronchial lymph nodes. The latter had many fibroblasts and collagen fibers in them.

Summary.—This 26-year-old patient developed a bronchopleural fistula twenty-eight days after a right pneumonectomy. This was preceded by a febrile period, and was followed by a sudden spread to the contralateral lung. At necropsy the oldest tuberculous disease was present in the right pleura. The only lesions in the left lung were of recent origin and were derived from the right pleural space.

CASE 3.—This 25-year-old white man became ill in October, 1944, when he developed an upper respiratory infection from which he did not completely recover. Pulmonary tuberculosis was discovered in December, 1944. Sputum was positive, and roentgenogram revealed a right upper lobe cavity with spread to the left upper lobe. A right pneumothorax was induced in February, 1945, but abandoned following thoracoscopy in April, 1945, because of pleural symphysis. Late in April of the same year a left pleural effusion developed and a cavity became evident in the fourth left anterior interspace. The effusion cleared, leaving an obliterated costophrenic sulcus. A left pneumothorax was attempted at this time, but abandoned. A right two-stage, five-rib thoracoplasty was done in November and December, 1945. Collapse was probably adequate. Sputum remained positive. A laryngitis which had the appearance of a typical tuberculous lesion occurred in December, 1946. He was given streptomycin (1.8 Gm. per day) from Dec. 9, 1946, to April 9, 1947 (217.8 Gm.). During this period he gained 10 pounds in weight, but his sputum was still occasionally positive. He was admitted to the Halloran VA Hospital in June, 1947.

Physical examination revealed a right-sided surgical collapse with some scoliosis. The right lung was clear to percussion and auscultation. There were some post-tussic inspiratory râles on the left side in the apex and posteriorly just medial to the scapula. Blood count, sedimentation rate, and urine examinations were all within normal limits. Sputum was Gaffky IX. Roentgenogram revealed a five-rib thoracoplasty with an open cavity under the major collapse. A 2 cm. cavity was present at the level of the left second rib anteriorly. There were scattered fibrotic and fibrocalcific nodules in the third left interspace.

A left phrenic crush was done in September, 1947. The left cavity enlarged during the next two months. The patient became febrile in January, 1948, and roentgenogram revealed a spread to the left mid-lung field. Further spread to the left lower lobe occurred in May, 1948. This did not clear with bed rest, and the patient again was placed on streptomycin (1 Gm. per day for forty days). A left extrapleural pneumothorax was done on Oct. 22, 1948. The cavity remained open. A small thoracoplasty (all of the second rib and diminishing segments of the third and fourth ribs) was done on Dec. 2, 1948. This in turn was followed by a segmental resection of the apical and posterior and anterior segments of the left upper lobe. The latter procedure was performed without incident.

Surgical Specimen.—Gross Examination: The specimen measured 11 cm. apicobasally, 8 cm. mediolaterally and 4 cm. anteroposteriorly. The covering visceral and parietal pleurae were fused, measuring from 3 to 5 mm. in thickness. The bronchi, including the proximal cut ends, appeared normal. A 4 cm. by 3 cm. by 1.5 cm. cavity was present in the basal aspects of the specimen. Its wall measured from 1 to 2 mm. in thickness. Occasional small encapsulated caseous and acinous nodose foci were present in the remaining portions of the lung parenchyma.

Microscopic Examination: Both acinous nodose foci and encapsulated foci were found to be in a stage of healing. The latter contained calcium deposits in the form of Liesegang rings. Many of the former consisted almost entirely of fibroblasts, collagen fibers, epithelioid cells, and lymphocytes, although some of the larger foci had small caseous centers. The thickened pleura consisted of dense collagenous connective tissue and contained large numbers of lymphocytes, plasma cells, and histiocytes. The segmental bronchus was free of foci, but contained a few lymphocytes, plasma cells, and histiocytes.

Postoperative Course: Streptomycin was given for forty-seven days (1 Gm. each day). It was necessary to leave a catheter in place for seven days. His temperature rose to 102° F. daily during the first eight days. He then ran a low-grade fever, his temperature reaching 100° F. On the twenty-third postoperative day he again began to spike a temperature which varied from 101° to 102° F. One month postoperatively his sputum increased in amount, and it became evident that a bronchopleural fistula was present. Closed catheter drainage was begun on Feb. 18, 1949. This did not control the situation, since spread of the disease to the left lower lobe was now noted. An anterior thoracoplasty was performed in April, 1949, and this area was packed three times weekly. His sputum was positive during this period of the postoperative course. The infecting bronchial closure. On February 23 the tenth rib was excised and the pleural space was packed with gauze soaked in streptomycin and penicillin. Roentgenogram at this time revealed massive spread to the left lung. He went into respiratory failure and succumbed on Mar. 11, 1950 (fifty-seventh postoperative day).

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CASE 3.—This 25-year-old white man became ill in October, 1944, when he developed an upper respiratory infection from which he did not completely recover. Pulmonary tuberculosis was discovered in December, 1944. Sputum was positive, and roentgenogram imperceptibly with that of the empyema space. A small segment of the latter was lined by stratified squamous epithelium which underwent transition to pseudo-stratified ciliated columnar epithelium when the bronchial lumen was entered. The distal surface of the bronchial cartilagenous plate was partially eroded by an extension of the tuberculous granulation tissue from the empyema space. There was no other tuberculous disease within the bronchial stump.

Summary.—This 26-year-old patient succumbed eight months after a segmental resection. His death was due to extensive bronchogenic spread of his disease through a bronchopleural fistula from a tuberculous empyema which developed following the resection. Postmortem examination indicates no other source for the spread than the empyema. The presence of stratified squamous epithelium in the lining of the empyema space adjacent to the fistula indicates that the fistula had been present for some time prior to death. The fistula was clinically apparent one month after surgery. It appeared following a febrile period during which purulent material accumulated in the dead space.

CASE 4.—This 30-year-old Asiatic-Indian man was discovered to have tuberculosis and leprosy in 1945. A right pneumothorax was started in 1946, but was abandoned in August, 1947. In 1948 he became febrile, lost weight, and had a small hemoptysis. His daily sputum production during this period was 60 to 120 c.c., and the sputum was positive for acid-fast bacilli. He was admitted to the Halloran VA Hospital on April 8, 1949.

The admission examination revealed him to be emaciated, febrile (101° F.), and acutely ill. Examination of the chest revealed dullness over the right chest posteriorly and in the right infraclavicular region. Râles were also noted in these regions. Posttussic râles were heard in right posterosuperior chest and infraclavicular region. The small muscles of the hand were atrophied and there was a left peroneal paresis. There was an erythematous eruption over the elbows, wrists, knees, and feet.

Laboratory data: Sputum was Gaffky VII on plain smear. Roentgenogram revealed a fibrocaseous involvement of the upper portions of both lung fields with multiple translucent shadows bilaterally. The right costophrenic angle was obliterated.

The patient became afebrile after a month of bed rest. Streptomycin (1 Gm. per day) was started on April 5, 1949, and was continued for forty-two days. Biopsy during this period revealed the skin lesion to be consistent with tuberculoid leprosy. On May 26, 1949 the patient had a hemoptysis. Streptomycin (1 Gm. every three days) with PAS (12 Gm. per day) was again started on July 15, 1949. The PAS was discontinued after five days because of nausea, but the streptomycin was continued until Oct. 16, 1949. The roentgenographic appearance of the lungs improved, with signs of cavitation disappearing on the left side. Hemoptysis assumed alarming proportions in September, however, and it was considered necessary to perform a right extrapleural pneumothorax on Sept. 30, 1949. This procedure stopped the hemorrhage, but the sputum remained positive. In December, 1949, hemoptysis recurred and it was necessary to give refills to maintain a highly positive extrapleural pressure. Since the patient had six months of stability on the left side, it was decided to perform a right pleuro-pneumonectomy. This was done on April 11, 1950. At operation the pleural space was obliterated and was not entered. There was extensive thickening posteriorly. The extrapleural space was clear. The bronchus was closed with end-on sutures, and the stump was covered with a pleural graft.

Pathologic Examination: A. Gross Appearance: The lung measured 21 cm. apicobasally, 6 cm. mediolaterally, and 12 cm. anteroposteriorly. Anteriorly the pleura was gray-white in color and measured less than 1 mm. in thickness. Posteriorly the fused pleurae measured from 3 to 8 mm. in thickness. A film of orange material could be peeled from the outer surface of the pleurae in this region. The lung cut with increased resistance in the apico-posterior aspects. The upper lobe was shrunken. Within the posterior aspects of the upper lobe was an irregular cavity measuring 2 by 2 by 1 cm. It was lined by a friable cheesy material. Its wall measured from 1 to 3 mm. in thickness and blended in one region with the adjacent pleura. A widely patent bronchus opened into this cavity. The remainder of the upper lobe parenchyma was firm, gray-white in organisms were sensitive to 2.5 μg of streptomycin prior to resection. The patient's condition was quite poor, so that fistula closure could not be attempted. On Aug. 6, 1949, he developed an upper respiratory infection which was followed by a severe tracheobronchitis. His pulse rate climbed to 140 to 200 per minute, and he developed evident pulmonary edema. He expired on Aug. 12, 1949, from pulmonary insufficiency.

Postmortem Examination: The left pleural space was obliterated in the lower two-thirds by loose adhesion bands (Fig. 3). In the upper third the visceral and parietal pleura were fused by dense gray-white tissue over all but the superior aspect of the lung. The superior surface of the upper lobe formed the base of a cavity which communicated, via a sinus tract, with the chest wall at the left anterior axillary line. Yellow pus filled this cavity. A small bronchus opened into the pleural cavity. Due to distortion of the pulmonary architecture, it could not be established which segment it originally served. Scattered throughout the left upper lobe and throughout both lower lobes were numerous acinous foci which varied from 3 to 5 mm. in diameter. These were more numerous and larger than in the resected specimen. No cavities were noted in any of the remaining lung parenchyma, although a dense radial scar in the upper lobe of the right lung probably represented a healed excavation.

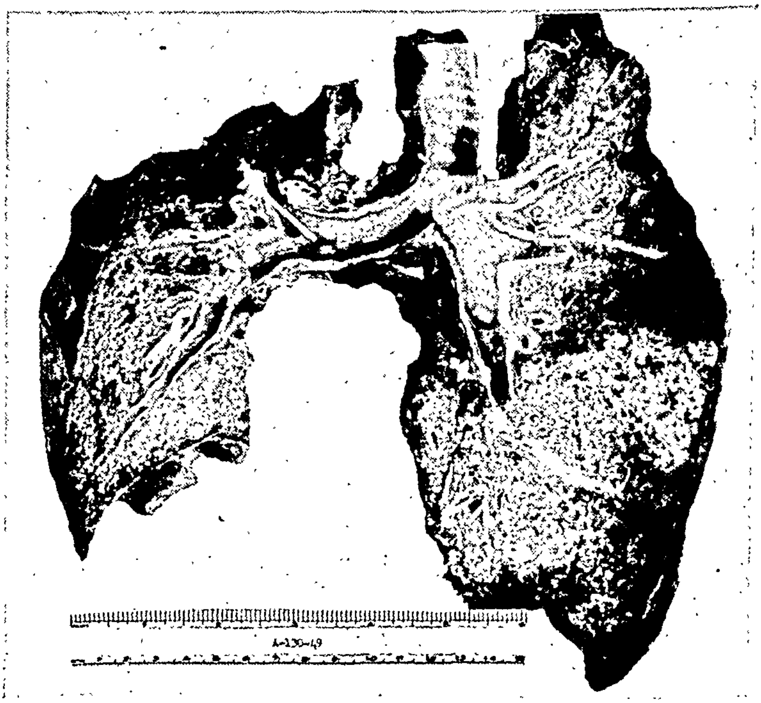

Fig. 3.—(Case 3) Tuberculous empyema following segmental resection. Probe is introduced into bronchus communicating with the pleural space. Note the large numbers of small foci of recent origin throughout the intact lung.

Microscopic Examination: Plugs of caseous material were found to fill the lumina of many bronchioles and were continuous with the central zone of caseation of adjacent acinous foci. The latter were surrounded by epithelioid cells, fibroblasts, and rare Langhans giant cells. The bulk of these foci were of far more recent duration than those observed within the resected specimen. The empyema space was lined by a wide zone of caseation. The underlying granulation tissue contained many epithelioid cells, Langhans giant cells, fibroblasts, and collagen fibers. Dense collagenous connective tissue formed the outer wall of the space. At the bronchocavitary junction the bronchial wall blended imperceptibly with that of the empyema space. A small segment of the latter was lined by stratified squamous epithelium which underwent transition to pseudo-stratified ciliated columnar epithelium when the bronchial lumen was entered. The distal surface of the bronchial cartilagenous plate was partially eroded by an extension of the tuberculous granulation tissue from the empyema space. There was no other tuberculous disease within the bronchial stump.

Summary.—This 26-year-old patient succumbed eight months after a segmental resection. His death was due to extensive bronchogenic spread of his disease through a bronchopleural fistula from a tuberculous empyema which developed following the resection. Postmortem examination indicates no other source for the spread than the empyema. The presence of stratified squamous epithelium in the lining of the empyema space adjacent to the fistula indicates that the fistula had been present for some time prior to death. The fistula was clinically apparent one month after surgery. It appeared following a febrile period during which purulent material accumulated in the dead space.

CASE 4.—This 30-year-old Asiatic-Indian man was discovered to have tuberculosis and leprosy in 1945. A right pneumothorax was started in 1946, but was abandoned in August, 1947. In 1948 he became febrile, lost weight, and had a small hemoptysis. His daily sputum production during this period was 60 to 120 c.c., and the sputum was positive for acid-fast bacilli. He was admitted to the Halloran VA Hospital on April 8, 1949.

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Laboratory data: Sputum was Gaffky VII on plain smear. Roentgenogram revealed a fibrocaseous involvement of the upper portions of both lung fields with multiple translucent shadows bilaterally. The right costophrenic angle was obliterated.

The patient became afebrile after a month of bed rest. Streptomycin (1 Gm. per day) was started on April 5, 1949, and was continued for forty-two days. Biopsy during this period revealed the skin lesion to be consistent with tuberculoid leprosy. On May 26, 1949 the patient had a hemoptysis. Streptomycin (1 Gm. every three days) with PAS (12 Gm. per day) was again started on July 15, 1949. The PAS was discontinued after five days because of nausea, but the streptomycin was continued until Oct. 16, 1949. The roentgenographic appearance of the lungs improved, with signs of cavitation disappearing on the left side. Hemoptysis assumed alarming proportions in September, however, and it was considered necessary to perform a right extrapleural pneumothorax on Sept. 30, 1949. This procedure stopped the hemorrhage, but the sputum remained positive. In December, 1949, hemoptysis recurred and it was necessary to give refills to maintain a highly positive extrapleural pressure. Since the patient had six months of stability on the left side, it was decided to perform a right pleuro-pneumonectomy. This was done on April 11, 1950. At operation the pleural space was obliterated and was not entered. There was extensive thickening posteriorly. The extrapleural space was clear. The bronchus was closed with end-on sutures, and the stump was covered with a pleural graft.

Pathologic Examination: A. Gross Appearance: The lung measured 21 cm. apicobasally, 6 cm. mediolaterally, and 12 cm. anteroposteriorly. Anteriorly the pleura was gray-white in color and measured less than 1 mm. in thickness. Posteriorly the fused pleurae measured from 3 to 8 mm. in thickness. A film of orange material could be peeled from the outer surface of the pleurae in this region. The lung cut with increased resistance in the apico-posterior aspects. The upper lobe was shrunken. Within the posterior aspects of the upper lobe was an irregular cavity measuring 2 by 2 by 1 cm. It was lined by a friable cheesy material. Its wall measured from 1 to 3 mm. in thickness and blended in one region with the adjacent pleura. A widely patent bronchus opened into this cavity. The remainder of the upper lobe parenchyma was firm, gray-white in color, and contained numerous encapsulated caseous foci measuring from 1 to 2 mm. in diameter. The middle lobe was resilient and well aerated. The lower lobe contained a few encapsulated caseous foci measuring 1 to 2 mm. in diameter. The intervening parenchyma was well aerated, and contained prominent alveolar sacs.

B. Histologic appearance: Neither the stem bronchus nor the peribronchial lymph nodes contained tuberculous lesions. The fused visceral and parietal pleurae were composed of dense connective tissue containing occasional lymphocytes and histiocytes laden with golden brown pigment. The surface lining the extrapleural space was covered by a thin layer of fibrin containing histiocytes laden with brown pigment and collagen fibers. There was no tuberculous involvement of any portion of the pleura. Advanced healing changes were noted about the upper lobe cavity and about the small encapsulated caseous foci in the upper and lower lobes of the lung.

Postoperative course: On May 8, 1950, a seven-rib thoracoplasty was performed. When the pleura was opened there was a small amount of fibrinous exudate on its surface. The pleural space contained thin fluid which was Gaffky I on smear and which was culture positive for tubercle bacilli. Culture for pyogens was negative. There were several 1 to 2 mm. raised yellow lesions over the lower mediastinal surface. No stump fistula was noted by the operator. A second stage was performed on May 18, 1950, removing segments of ribs eight and nine. Fluid removed at this procedure was negative. The pleural space was tapped on June 8, 1950, and a cloudy fluid was removed. This was also negative for acid-fast bacilli. Methylene blue was injected at this time, but was not expectorated. The patient ran a low-grade fever, but chest roentgenograms showed no further changes. On June 16, 1950, the right chest was again tapped and was positive for acid-fast bacilli. Methylene blue injected at this time was expectorated, confirming the presence of a bronchopleural fistula. All efforts to close the fistula were fruitless, and the patient died from an overwhelming spread to the intact lung in November, 1950.

At necropsy the intact lung was found to contain large numbers of caseous lobular pneumonic areas and acinous nodose foci (Fig. 4). Lack of healing changes in and about these foci indicated that they were of recent origin. In addition there were numerous smooth-lined, thin-walled sacs within the upper lobe. These had a ramifying structure and were lined in part by a thin layer of yellow cheesy material. These were interpreted as recent tuberculous ulcers in ectatic bronchi, since the smooth lined portions of these structures were lined by tall pseudo-stratified ciliated columnar epithelium. There were no thick-walled cavities or other evidence of old excavations which might have served as a source of infection of the pleural space.

The right pleural space was filled with thick yellow pus which was culture positive for *M. tuberculosis.* The residual space measured 17 cm. vertically and 5 cm. mediolaterally. It was lined by thick yellow material. Deep to this was a thick layer of dense gray tissue. A 1 cm. segment of the right main bronchus remained in situ. Its mucosa was red granular in appearance. A 4 cm. opening at the distal end of the stump communicated with the dead space.

In summary this 30-year-old patient developed a bronchopleural fistula sixty-six days following right pleuro-pneumonectomy. The residual chest space was culture positive for tubercle bacilli, and yellow foci were noted on the pleural surface one month after the resection. No stump fistula was seen at this time. All of the lesions in the intact lung were of recent origin, and no older lesions were noted which could have infected the pleural space.

CASE 5.—This 23-year-old Negro was found to have tuberculosis in March, 1946. Roentgenogram revealed bilateral upper lobe cavities with an extensive exudative component in the left upper lobe. Sputum was positive for acid-fast bacilli. He was put on bed rest. After one year he became febrile and began to lose weight. In May, 1947, streptomycin (1 Gm. each day) was started and continued for one hundred twenty days. He was transferred to Halloran Veterans Administration Hospital on June 23, 1947.

Examination revealed him to be moderately emaciated. Chest examination revealed a lag on the left. There was increased tactile fremitus over the left upper lobe. Bronchovesicular breath sounds and a few moist râles were present in the same area. The right side was clear. Sputum was positive for acid-fast bacilli on direct smear. Roentgenogram revealed that the upper two-thirds of both lung fields were involved by a fibrocaseous infiltrate with numerous areas of radiolucency. The trachea was shifted to the left.

The patient was continued on streptomycin. The right side cleared well, but he was left with a fibrocaseous disease largely replacing the left upper lobe. Sputum remained positive (Gaffky VII). A six-rib two-stage left thoracoplasty was performed in December, 1947, and January, 1948. Apicolysis was performed at the same time. Although it was a technically good thoracoplasty, the sputum remained positive. Streptomycin was again given from July to September, 1948 (54 Gm.). Streptomycin sensitivity studies at this time revealed the infecting organisms to be sensitive to 2.5 μg per cubic centimeter. On April 22, 1949, a left upper lobectomy was performed. There were dense pleural adhesions between the parietal and visceral pleura over the upper lobe. A cavity in the apicoposterior segment was entered in the course of freeing the lobe under the thoracoplasty.

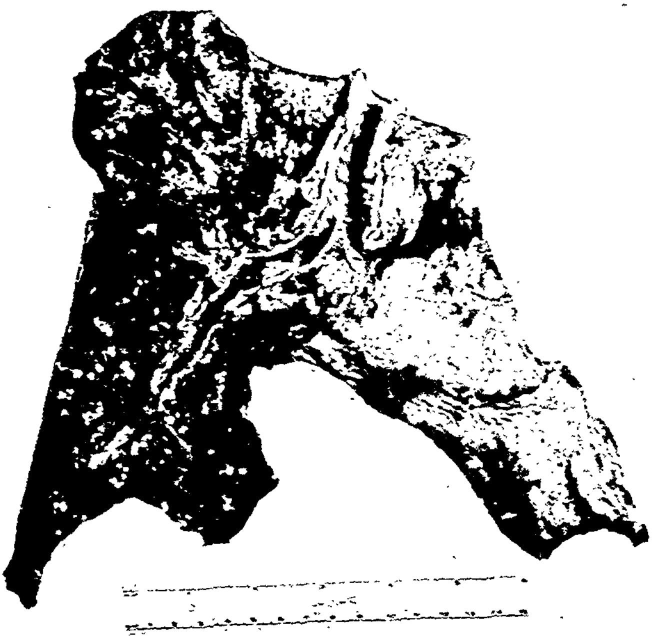

Fig. 4.—(Case 4) Tuberculous empyema, widely patent stump fistula, and many fresh lesions in intact lung parenchyma.

Pathologic Examination of the Resected Specimen: Gross: The compressed upper lobe of the left lung measured 13 cm. apico-basally, 6 cm. mediolaterally, and 2.5 cm. anteroposteriorly. The lobe cut with increased resistance. A cavity measuring 3 by 2.5 by 2 cm. was present in the lateral, apical, and posterior aspects. This was lined by a gray-yellow cheesy material. Its wall measured from 1 to 4 mm. in thickness. The lumen of this cavity communicated with the pleural surface by way of an irregular opening (surgical artefact already described). The remainder of the lobe contained several encapsulated caseous foci which measured from 1 to 2 mm. in diameter. The walls of the upper lobe bronchi had a granular appearance, but no gross ulcerations were noted. The pleura was smooth and shiny in all but the apico-posterior aspects where this segment of the lobe was dissected free from the chest wall.

Microscopic Examination: The only tuberculous involvement of the upper lobe bronchi noted was an ulcer at the bronchocavitary junction. Advanced healing changes were noted in the wall of the cavity and about the smaller foci within the lung. The pleura was without evidence of tuberculous involvement.

The patient's postoperative course was stormy. He was febrile for thirty-three days and developed a pleural effusion which never completely cleared. Unfortunately this fluid was not cultured. His sputum, except for a Gaffky I, was negative until September, 1949 (approximately one hundred thirty days), when his sputum then suddenly increased in amount and became strongly positive. All roentgenographic techniques failed to implicate the left lower lobe as the source of these organisms, but in view of the stable right lung, it was felt that the left side must contain an active focus. Bronchoscopy on Dec. 6, 1949, failed to reveal definite evidence of disease, but the mucosa at the level of the superior segmental orifice was edematous and reddened. Sputum was Gaffky VI in February, 1950. Streptomycin sensitivity was less than 10 μg per cubic centimeter. Streptomycin and PAS were started on Jan. 5, 1950, and were continued for ninety days. There was an episode of streaking in late January, 1950. On February 21 a left lower lobectomy was performed with considerable difficulty. There was an empyema space overlying the lobe and the stump of the upper lobe bronchus. The latter was covered with cheesy yellow material. There was an area just above the superior segment of the lower lobe which was very close to the bronchial stump which contained semifluid purulent material. There was also a large plaque of caseating tuberculosis over the pericardium. All caseous areas were removed with the specimen. Culture of this area revealed the presence of acid-fast bacilli but no pyogens.

Pathologic examination: The lower lobe measured 9 cm. apico-basally. The lung parenchyma was resilient and well aerated. It contained a few encapsulated caseous foci which measured from 1 to 3 mm. in diameter and a few small acinous foci. There were no areas of caseous lobular pneumonia or cavities within this portion of the lung. The pleura and the adjoining pericardium were lined by a layer of nodular gray tissue which was covered in areas by a layer of cheesy yellow material. The upper lobe bronchial stump was not available for examination. The peribronchial lymph nodes were slate gray black in color.

Microscopic appearance: The encapsulated and acinous foci in the lower lobe were in an advanced state of healing. Large numbers of small foci in an advanced state of healing were present in the regional lymph nodes. The pleura and adjacent pericardium were covered by a wide layer of caseous material. Beyond this was a pyogenic membrane composed of fibrin and polys. A wide granulation tissue composed of fibroblasts, epithelioid cells, Langhans giant cells, collagen fibers and small capillaries underlay the pyogenic membrane. Small round tuberculous foci were present within this granulation tissue. The pleura beyond this was greatly thickened by dense collagenous connective tissue.

In summary, it is felt that this patient developed a tuberculous empyema followed by a bronchopleural fistula, the latter having been the source of the acid-fast organisms which appeared in the sputum approximately one hundred thirty days after the left upper lobectomy. This assumption is supported by the fact that no source of these organisms could be demonstrated in the lower lobe, and since no organisms have been found in the patient's sputum or gastric contents since the second operation (eight months). Treatment has been effectual in clearing the pleural disease and closing the dead space. The patient has been discharged from this hospital.

DISCUSSION

For purposes of discussion we may divide the possible causes of postoperative fistula formation in tuberculous patients into several categories. We believe that it is possible that, in the future, instances of any one of them may be observed, although it seems improbable that some of these mechanisms have a great importance in this regard.

A. Erosion by an Extrinsic Lesion.—

1. *Tuberculous empyema:* Erosion of a bronchial stump by tuberculous empyema would enable the escape of infected material from the empyema space into the tracheobronchial tree, with subsequent spread of the disease to intact pulmonary tissue. This has been the commonest cause of fistula formation in our series, and we feel that it was operative in all of the cases presented. To establish this mechanism as the cause of these fistulas we must furnish evidence that the tuberculous empyema existed prior to fistula formation, and that no other tuberculous lesions existed that were capable of infecting the pleural space. These criteria are fulfilled in each of the five cases presented, although in some instances the evidence is indirect. In each of the first three autopsy cases the pleural lesion was the oldest active tuberculous lesion in the pulmonary system, and the only ulcerocavitary disease present. Such lesions as were present in the intact lung were small and were either inactive (radial scars and encapsulated caseous foci) or the result of a spread from the pleural space (recent acinous nodose foci). In the living patient (Case 5) no cavities were demonstrated in the nonexcised parenchyma prior to surgery either by standard radiographic techniques or by tomography. Such cavities as were noted in the contralateral lung (Case 4) appeared subsequent to fistula formation and represented infection of bronchiectatic sacs from the empyema space. We believe this to be true because the cavities were thin walled, followed the ramifications of the bronchi, and were not visible by roentgenogram until after the formation of the stump fistula. Bacteriologic evidence that the tuberculous empyema existed prior to fistula formation was present in Cases 1 and 4. Histologic evidence substantiated the diagnosis in Case 5. In Cases 2 and 3 a tuberculous empyema was present at necropsy, and there was no source of tuberculosis in the remaining lung parenchyma which could have infected the space after fistula formation.

It may be asked whether it is possible for a tuberculous empyema to erode a bronchus, in the face of evidence that extension into the lung parenchyma by tuberculous empyema almost never occurs.[1] It is of interest in this regard that we have seen a nonsurgical case in which the trachea was eroded by tuberculosis of the adjoining pleura. Similar erosion of the bronchial stump seems possible in the light of this observation, since the traumatized bronchial tissue would furnish less resistance to an advancing ulcerative process than would an intact trachea.

It is noteworthy that the fistulas in these cases occurred at forty-one days, twenty-eight days, thirty days, sixty-six days, and one hundred and thirty days after excisional surgery. We feel that this time lag is more consistent with the indolent course of tuberculous processes than with errors in surgical technique. Fistulas of the latter type usually appear during the first fourteen days of the postoperative period. We have found that fistula formation is often preceded by a febrile episode during which either the chest fluid or samples of the pleural tissue give evidence of tuberculous activity. We feel that any patient developing unexplained fever in the postoperative period should be studied to rule out tuberculous involvement of the pleural space. In the event that such infection exists, every effort should be made to prevent the development of a fistula. These views parallel those stated by Kent[3] and Day and associates.[4] The former author makes the additional observation that late fistulas occur as a result of erosion by a nontuberculous empyema. We have one such case in our series of nontuberculous resections. It has been our experience, however, that there appears to be a much greater likelihood of erosion in the presence of tuberculous infection.

It remains to establish the cause of the empyema. It may be derived from contamination of the pleural space at the time of surgery. Accidentally opened pulmonary cavities or incision of frankly caseous hilar lymph nodes could serve as sources of infection. In only one of our cases (Case 5) was a cavity opened. Pus may also escape from the raw pulmonary surface by way of the alveoli and small bronchi and bronchioles. This could occur during segmental resections, or during difficult cases where the visceral pleura over the diseased lung is torn free of the pulmonary surface. One of our cases (Case 3) was a segmental resection and may perhaps have fallen into this group. Opportunity for the escape of infected secretions into the pleural space may occur when the stem bronchus is cut. Last, the empyema may represent an exacerbation or continuation of an infection existing prior to surgery. In our series, despite careful search, none of the surgical specimens showed any evidence of tuberculous involvement of the pleura. However, it is possible that small unsuspected caseous foci may have been left in situ at the time of surgery. Since pleural tuberculosis is often of hematogenous origin from an extrapulmonary source[2] it is of passing interest that two of these cases (Case 1 and 3) had genitourinary tuberculosis. It is possible that unsuspected hematogenous pleural tuberculosis existed in these cases, and that this served as a source for the subsequent empyema.

The objection might be raised that surgical contamination probably occurs more frequently than does postoperative tuberculous empyema. It is difficult to estimate the amount of contamination in any one case. Individual resistance and degree of dosage are the determining factors in any infection, and in this instance neither of these is readily measurable. Another factor which may possibly play a role in determining whether a given spill will result in a tuberculous empyema rests with the growth characteristics of the invading organism. Although the pleural dead space may furnish an ideal container for the culture of bacterial organisms, there are individual variations in the culture medium. Sensitive organisms are adversely affected in the presence of streptomycin. Hurwitz,[5] however, has shown by in vitro methods that the growth of resistant organisms is stimulated by the presence of streptomycin in a medium which varies from pH5.8 to pH6.2. This effect is most pronounced from pH6 to pH6.2. In some cases he has noted that the organisms will not grow unless streptomycin is present. It would be interesting if pH studies of pleural aspirates were combined with sensitivity studies of the infecting organisms to determine whether a similar relationship exists within the postoperative chest space.

2. *Caseous tuberculosis of the hilar lymph nodes:* Rupture of a caseous lymph node into an adjacent bronchial stump is a possible cause of fistula formation. Intact bronchi are occasionally eroded by disease extending into them from such nodes, but this is a relatively uncommon phenomenon. It does not seem likely that this might be a frequent occurrence, since it usually occurs in patients with extensive lymphohematogenous tuberculosis, and these patients are not likely to come to surgery. Usually the lymph nodes in chronic pulmonary tuberculosis are void of all but a few microscopic-sized miliary foci.

B. *Intrinsic Causes of Stump Fistula.*—

1. *Improper closure of the bronchial stump:* Technical errors must be the source of some fistulas. The frequency with which this complication is reported following excisional surgery for bronchogenic carcinoma and for other nontuberculous pulmonary lesions gives substance to this belief. It is not the purpose of this paper to catalogue all of the sources of error, but rather to assess the importance of such errors as causative factors in fistula formation. It would be surprising if errors were not committed in the treatment of tuberculosis, since the technical difficulties encountered in the surgical therapy for this condition may be equal to or greater than those attending carcinoma or bronchiectasis.

Following slough of the stump a mixed pyogenic empyema can then supervene. In the absence of a source of tuberculous infection, the empyema will remain of this type. This is an important point in distinguishing this form of fistula from one developing on the basis of an erosion by a tuberculous infection of the dead space. If, however, the pleural space has been contaminated by caseous material, or if a cavity exists in the remaining lung parenchyma which can infect the space through the fistula, it may present a clinical appearance similar to that seen following extension of a tuberculous empyema. In this case we feel that the time of onset of the fistula should be borne in mind. It is our opinion that fistulas occurring during the first two postoperative weeks should be placed in this category, since time allowances must be made for the development and progression of the tuberculous infection within the pleural space. It should also be noted that it is often difficult to give a precise date of onset in patients of this group, since the febrile episode which attends the empyema and fistula often blends with that which follows surgery. The fistulas of two patients in this series should be placed in this group. The fistulas occurred on the fifth and tenth postoperative days following segmental resection. In each case the sputum was negative for tubercle bacilli on smear and culture despite the presence of the fistulas. The fluid within the empyema space was negative for tubercle bacilli and positive for pyogens. Both of these fistulas were subsequently closed and the patients are free of active tuberculosis.

Kent[3] stated a similar concept in these words: "It is my opinion that a bronchial fistula which occurs in the first ten days after operation is a true failure of healing of the bronchial stump and that the associated empyema is of secondary origin. On the other hand, in those instances of a later leak in the bronchus, usually from three to five weeks after pneumonectomy, the cause is most commonly infection in the pleural cavity with an empyema which escapes through the recently healed bronchus." Kent's views were derived from a study of bronchial fistulas in thirteen nontuberculous patients. Of these patients, six occurred in the first ten postoperative days, and seven were in the later group. Among seventy-eight cases of excisional therapy for nontuberculous conditions, we have encountered early fistulas in four instances. One of these four patients died on the twelfth postoperative day. The remainder followed a course similar to that observed in the two tuberculous patients already noted in this group.

There exists one technical factor which may have a bearing on the ability of bronchial tissue to resist a progressing ulcerative lesion within the pleural space. In each of the three pneumonectomies which came to necropsy, it was noted that the bronchial stump measured 1 cm. in length. It is possible that a blind sac of this size may retain sufficient infected secretions to alter local tissue resistance. It would be of interest in the future if the length of the stump were to be calculated in all cases coming to necropsy so that this factor could be evaluated.

2. *Extension of a tuberculous ulcer of the bronchus through the stump:* Tuberculous ulcers of the bronchi are, with a few exceptions, found only in the presence of cavitary disease of the lung. If a pulmonary cavity closes, or is resected, it is likely that the bronchial ulcer will heal. Therefore, it does not appear probable that such an ulcer would go on to progression after resection unless the bronchi were more severely involved than usual. If the entire thickness of the bronchial wall had undergone caseous necrosis, no technique of closure would prevent a slough with subsequent fistula formation. Bronchial disease of this degree is relatively rare, however. In any case there was no evidence that this mechanism was operative in any of the cases reported here, since histologic examination of the proximal edges of the resected bronchi did not reveal any evidence of tuberculosis. On the other hand, when tuberculous lesions are discovered in the stumps of resected lungs they are usually small submucosal foci. None of the patients in whom we have noted these foci have developed fistulas. Day and associates[4] offer additional support to this statement. They say that, in their series, cases have been noted in which adequate healing occurred when transection was carried through grossly diseased mucosa.

3. *Infection of the bronchial stump by a tuberculous cavity in the intact lung:* Too little necropsy material has accumulated to judge whether this mode of stump infection could occur. We do not believe that it was a cause of any of the cases reported herein, although we have only clinical and radiologic evidence to support this statement in the living patient (Case 5).

SUMMARY

1. Five tuberculous patients, who developed bronchopleural fistulas during the postoperative period as a result of excisional therapy, were studied with a view to establishing the pathogenesis of the fistulas.

2. Of the five patients, four died and were studied at necropsy, whereas one remains well.

3. Massive involvement of the intact lung by recent tuberculous lesions was felt to be the immediate cause of death in the fatal cases. It was felt that in each instance a tuberculous infection was derived from the infected pleural space.

4. It is felt that the principal causes of post excisional fistulas are extension of a tuberculous empyema through the bronchial stump and technical errors in stump closure. All of the tuberculous patients in whom this complication was noted fall into the first category. Comparison of this group of patients with similar patients in the nontuberculous series revealed that the fistulas occurred late in the postoperative course in the former group and tended to occur within the first fourteen postoperative days in the latter group. It is assumed that early fistulas in nontuberculous patients are due to technical errors in stump closure.

5. It is felt that the fistulas in the tuberculous patients occurred as a result of extension from a tuberculous empyema because the empyema preceded the fistula in three of the cases. In two other cases no source of pleural infection existed in the intact lung.

6. It is felt that the tuberculous empyema results from contamination of the pleural space at the time of operation, although this factor is difficult to evaluate in the individual case.

7. It is possible that an excessively long bronchial stump may harbor infected secretions which may alter the resistance of the bronchial tissue to a progressing tuberculous empyema. A greater number of autopsied cases is required to establish this as an important factor.

8. As in nontuberculous cases, early fistulas are thought to be due to failure in stump healing. If no source of acid fast bacilli exists in the intact lung to infect the pleural space, the resulting empyema is pyogenic in character. Such fistulas usually pursue a benign course. Two cases of this type were noted in this series.

9. It is felt that neither tuberculous ulcers of the bronchial stump nor hilar lymph node tuberculosis is an important source of bronchopleural fistula.

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DISCUSSION

DR. JAMES D. MURPHY, Oteen, N. C.—Having had our share of these catastrophies, sometimes I think more than our share, we have been extremely interested in the problem of bronchopleural fistula following resection for tuberculosis. Since Dr. Auerbach told us a year ago that he had evidence indicating that the cause of the fistula could usually be traced to an empyema, we have been waiting to hear the completed paper.

It is pleasing to note that his findings almost parallel our own. In a recent report we studied autopsy findings in five patients who died following a fistula. These five patients were compared to a group of twenty-two who developed postresection fistula and with our total series of resections. We found that in the five autopsied patients the bronchial stump was literally eaten away by tuberculous granulation tissue. The three groups were analyzed from every angle that we could think of. Since we could find no difference between the technical aspects of the surgery, pre- and postoperative care of the selection of cases were considered. In only one factor was there a definite trend established, and that was in the use of streptomycin. Nowadays everyone receives streptomycin long before they come to surgery. In order to obtain statistics resulting from a large series of cases, we pooled our own contribution of 186 resections with the entire series reported at the Tenth Veterans Administration Streptomycin Conference in Atlanta in January, 1951. I would like to present those results to show the deleterious effect of the previous streptomycin treatment.

(Slide.) This represents 476 resections who came to the surgeon having had previous courses of streptomycin therapy and 480 resections who had no previous streptomycin prior to their surgical prophylactic course. All of these patients were treated with streptomycin for one week before and two weeks after resection. Fistula developed in 13.2 per cent of those previously treated and only 5.3 per cent of those who had antibiotic treatment prior to the resection course.

(Slide.) Further analysis was made of those who were known to be sensitive and those who were known to be resistant at the time of resection. Fistula developed in 26.8 per cent of ninety-seven patients who were known to be resistant to streptomycin at the time of surgery. Only 10.7 per cent of the sensitive patients developed fistula. These figures were collected from a group of forty-six participating Veterans Administration study units. The surgery in these hospitals is supervised for the most part by members of this Association, so we will have to accept the fact that it was good surgery.

(Slide.) This represents our own troubles at Oteen in a group of patients in whom a resection was done for one definite indication; a residual post-thoracoplasty cavity. Thirty-nine successive patients who had no previous streptomycin treatment were resected for this indication without a fistula. In twenty-three post-thoracoplasty resections who had had one or more courses of streptomycin before coming to surgery, seven patients, or 30.4 per cent, developed a bronchopleural fistula. The relationship of previous streptomycin treatment to complications following surgery has been studied in other forms of the surgery of tuberculosis. The results following thoracoplasty and those following cavernostomy for residual cavity also indicated that previous treatment with streptomycin resulted in an increase in the number of postoperative complications.

DR. C. THOMAS READ, Phoenix, Ariz.—Recently in the literature references and articles have appeared concerning the use of streptokinase-streptodornase outside the original reporting group in New York. With the increased use of the enzymes, I would like to call attention to some of the data we accumulated and reports obtained from others regarding bronchopleural fistula. We believe bronchopleural fistula does not occur as a direct result of the use of the material, but follows more or less indirectly. At the meeting last year it was mentioned that fistulae or potential fistulae may be controlled or made dormant by the tamponade effect of fibrinous debris. Streptokinase-streptodornase are fibrinolytic and possess the unique qualities of dissolving nucleoprotein, and they therefore could remove the tampon effect mentioned. In pulmonary resection cases it is well to keep these points in mind if indications arise for the use of the enzymes.

DR. HERBERT C. MAIER, New York, N. Y.—I think a word might be said to try to clarify some of the confusion as to the incidence of bronchial fistula in relation to streptomycin therapy. It would seem that the best results in so far as avoidance of fistula is concerned occur either when excisional surgery is done with the first course of streptomyci [illegible] bacilli are still streptomycin-sensitive, or after a very prolonged uninterrup [illegible] reptomycin combined with PAS. It seems to me that the highest incidence of fistula occurs where an intermediate amount of streptomycin has been used in several short courses. Then one has lost some of the beneficial effect of the streptomycin and bacterial resistance may have developed, but one has not yet obtained the maximum subsidence of the tuberculous process. This is particularly important if bronchial disease was present close to the line of section of the bronchus. Therefore, if you cannot or do not consider it advisable to do the excision with the first course, it is best to wait some months for the long course of streptomycin and PAS, in order to have maximum subsidence of the tuberculous process in the bronchus and elsewhere at the time of excision.

DR. IRVING A. SAROT, New York, N. Y.—The studies of Dr. Stemmermann and his colleagues provide a valuable confirmation of the published views of Tchertkoff and Selikoff that some postpneumonectomy bronchopleural fistulae occur as a result rather than as a causative factor of postoperative empyema. It has been in an effort to reduce the incidence of such empyemas that removal of the parietal pleura has been recommended in the course of all pneumonectomies for tuberculosis, not only because bits of tuberculous plura and involved lung remaining after a difficult intrapleural dissection may be the source of the empyema, but also because the parietal pleura becomes infected easily as a result of contamination during operation.

(Slide.) Extrapleural pneumonectomy (pleuropneumonectomy) was developed to overcome the difficulties in resection occasioned by the presence of dense pleural adhesions (symphysis) or empyema. This procedure reduces the incidence of contamination of the operative field due to rupture of lung, cavity, or empyema pocket, and also removes the parietal pleura which reacts to infection so vigorously by exudation and caseation. All the parietal pleura should be removed. Total removal of the diaphragmatic pleura, while most desirable, may be difficult due to the anatomic peculiarity of the underlying connective tissues which make for more intimate attachment of the pleura in this region. When, inadvertently or unavoidably, significant portions of the costal or mediastinal pleura are not removed, the incidence of empyema will increase. Surgical exploration of a number of cases of empyema following pneumonectomies considered to have been done extrapleurally has revealed the presence of residual parietal pleura.

Study of a large series of consecutive pneumonectomies performed by the various surgeons on the staff of Dr. L. R. Davidson at Sea View Hospital revealed significant differences in the postoperative results of intra- and extrapleural pneumonectomies.

(Slide.) Of 118 consecutive pneumonectomies for tuberculosis without empyema, seventy-five were done intrapleurally and forty-three extrapleurally. Among the seventy-five intrapleural pneumonectomies, in which the parietal pleura was permitted largely to remain in situ, there were thirteen postoperative empyemas with eleven deaths. Most of these empyemas were associated with bronchopleural fistulae, and clinical evidence was clear that in many of the cases the empyema preceded the opening of the bronchial stump. In the forty-three extrapleural pneumonectomies in this series, there were no postoperative empyemas, despite obvious gross contamination from rupture of a cavity at operation in four cases.

Therefore, especially as it has no indispensable function after pneumonectomy, it seems proper to remove the parietal pleura in every pneumonectomy for tuberculosis, as the incidence of postoperative empyema and bronchopleural fistula is thereby reduced. This is not to say that pleuropneumonectomy is not occasionally followed by either a bronchopleural fistula or empyema, or both, since they do occur although much less frequently; but they manifest a less toxic course.

(Slide.) The extrapleural dissection which circumvents dense adhesions and matted hilar nodes facilitates a rapid approach to the carina and permits a high amputation of the bronchus with resultant short bronchial stump. In two cases, Dr. Stemmermann noted at autopsy bronchial stumps 1 cm. in length. Such long stumps are prevented from retracting into the mediastinum by adherent peribronchial and pleural connective tissues. They are then more liable to open primarily because of failure of surrounding tissues to cover them, or to be eroded secondarily by empyema.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—Very little has been said in this discussion about the manner in which the bronchus is closed. It has a bearing on the incidence of empyema. I am sure that almost everyone here has his own pet method of effecting a satisfactory closure of the bronchus. I have personally come to the conclusion that mattress sutures should be avoided and that something should always be done to the cartilage in order to prevent tension on end sutures. A radical change in the type of bronchial closure was made two years ago and in 228 consecutive bronchial closures there has been just one fistula.

(Slide.) I would like to illustrate this simple method. Possibly some of you use it. No clamps are used on the proximal side of the bronchus. The bronchus is only partially divided at first. The attached lung and bronchus are used for traction. Two cartilages are cut at their center from within. The first suture is passed through the end of the bronchus about midway between the membranous portion and the point of fracture of the cartilage, then out and in through the membranous wall to the opposite side of the bronchus. When this suture is set, the membranous wall becomes inverted to reduce the bronchial width by half. A second suture is placed above and a third below the first. Usually three end sutures are sufficient. The use of a minimal number of sutures prevents devascularization of the stump and will do more to prevent difficulties with healing and infection of the space than any other single step.

DR. ROBERT M. JANES, Toronto, Ontario.—After hearing Dr. Overholt's discussion I felt that it was probably up to me to make what might be considered a confession, because we have always believed in the simplest possible closure of bronchial stumps and we still use a simple running suture of chromic catgut, being careful to apply it so that it does not produce tension and interfere with nutrition of the end of the stump. I think that the important factors are the same as in surgery of the bowel, surgery of the colon, the least possible trauma, and closure without tension on the tissues and without tension on the sutures.

I have been most interested in the bringing forward of these problems. It seems to suggest thought along other directions than methods of complicated closure which I am quite sure are not the answer to this problem.

DR. STEMMERMANN (closing).—I want to thank the discussors. First, with regard to streptomycin, I do think there is one interesting factor that should be emphasized. Environmental factors may affect the growth characteristics of the invading organism, streptomycin adversely affecting organisms sensitive to the drug. It is interesting, however, that Hurwitz has shown by in vitro methods that the growth of resistant organisms is stimulated by the presence of streptomycin in a culture medium which varies from pH 5.8 to 6.2, the effect being most pronounced from pH 6.0 to pH 6.2. In some cases he has actually noted that the organism will not grow unless streptomycin is present under these conditions. It would be interesting in the future if pH studies of pleural aspirates were combined with sensitivity studies to determine whether a similar relationship exists within the chest fluid. Types of technical errors were not elaborated, since we wished only to assess the importance of errors in the pathogenesis of fistulas.

BRONCHIECTASIS: A COMPARATIVE STUDY OF TUBERCULOUS AND NONTUBERCULOUS PYOGENIC SUPPURATIVE DISEASE

Joseph Gordon, M.D., Ray Brook, N. Y., and Philip C. Pratt, M.D.
(by invitation), Saranac Lake, N. Y.

THIS presentation of a comparison between tuberculous and nontuberculous bronchiectasis will be limited to four points. In the formal completed manuscript other data will be considered. The term bronchiectasis is an unfortunate one, since by definition it means only dilatation of the bronchial tubes, but by inference and common usage has come to mean a disease entity. Nontuberculous bronchiectasis as used in this instance is that disease entity which commonly appears in young children and continues through later years, usually associated with cough and expectoration of large amounts of purulent foul smelling and tasting sputum. This, of course, is the type of disease that is generally meant when the word bronchiectasis is used, without other qualifying terms. Tuberculous bronchiectasis commonly means an associated disease of the bronchi in a patient with pulmonary tuberculosis. Often no distinction is made as to the level of the order of the bronchial tree that may be implicated. However, this immediately becomes clear when bronchograms are done and the abnormal dilatations are noted. This, then, gives a reference point when it is used in tuberculosis as in nontuberculous bronchograms.

The points of comparison that are being here made and illustrated in the slides are as follows:

1. Segmental lobar location of tuberculosis versus nontuberculous bronchiectasis. That either of these two diseases may involve an entire lung or may be present on both sides of the chest is recognized. Tuberculosis, as is well known, is commonly located in the posterior and apical portions of the lung segments. In contrast to this, nontuberculous bronchiectasis is commonly located in the posterior and basilar portions of the lung. In addition, tuberculosis in the apical posterior portions of the upper lobe is not uncommonly associated with disease in the superior segment of the lower lobes. This has been of more recent appreciation during exploration at the time of pulmonary resection for tuberculosis. There is a similar parallelism in nontuberculous bronchiectasis in that disease located in the posterior portions of the lower lobes often has contiguous upper lobe involvement in the lingula on the left and middle lobe on the right. In addition, in recent years, a clinical syndrome has been recognized of pneumonitis with bronchiectasis involving the right middle lobe alone.

2. Bronchograms. Tuberculous and nontuberculous cases show some similarities in that dilatation of the bronchi are thereby depicted. However, by

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direct comparison, whether in the presence or absence of cavitary disease in tuberculosis, two differential points have been noted. These are that in the tuberculous case the bronchograms show evidence of dilatation interpreted as bronchiectasis, but the filling is in the medial two-thirds of the lung field and *distinctly absent from the outer portion of the lung field.* In nontuberculous bronchiectasis the distinguishing characteristic is that, while a smaller sized lobe than normal is seen, the dilated bronchi extend to within the visceral pleural margin of the lobe. This has not been seen in tuberculosis.

3. A further point, as recognized in the bronchogram films, is that when these films are taken in both inspiration and expiration one sees little, if any, change in caliber of the dilated bronchi in nontuberculous cases, whereas in the tuberculosis cases one notes relatively normal behavior of the bronchi, for in expiration they are seen to decrease in size.

4. A fourth point of difference observed in this study between tuberculous and nontuberculous bronchiectasis has been observed in a study of films after injections of surgical and postmortem specimens with iodized oil. The contrast medium was injected into the bronchi and pulmonary arteries. In pulmonary tuberculosis one sees, as is well known, evidence of obliterative arteritis, and this same obliterative change is seen in the bronchi as well. Again dilatation of the larger bronchi is seen with shortening, widening, and lack of arborization, in addition to the fact that the abnormal shadow is seen to stop well within the inner portions of the lung and to be absent from the outer third. This is in striking contrast to findings in nontuberculous bronchiectasis studied *in the same manner. Here one sees most strikingly that the pulmonary* arterial system is completely intact, that the bronchi have the same widened appearance as noted in bronchograms with irregular margins and few or no branches, and extend to within the pleural margin of the lobe. These two distinct patterns, as above described, have been repeatedly demonstrated in all cases of bronchiectasis studied thus far, and are a constant finding in tuberculosis.

When one reviews the histopathology in the two diseases, it is easy to understand how these two patterns are demonstrable in the roentgenograms of injected specimens. The commonly seen necrotic destructive lesion of tuberculosis involves all elements of the lung parenchyma, including the bronchi and pulmonary arteries, or in some areas dense scar tissue replacement is seen to efface the smaller arterial branches as well as the bronchi and bronchioles. The bronchi toward the hilum are seen to be uninvolved, as the entire structural element appears intact. There are individual exceptions which are readily recognized. One is specific tuberculous inflammation or ulceration followed by stenosis of a major bronchus, and here the picture is that of narrowing of the lumen associated with some distal dilatation, but this is not the pattern here referred to as bronchiectasis. The pattern of bronchiectasis as described in the roentgenogram, following the use of iodized oil, is the most common one and, while these bronchi are seen to go to the lobe or portion of the lung that is extensively involved, they themselves appear intact and uninvolved by the specific disease.

A study of the histopathology of bronchiectasis which has been done repeatedly, carefully, and by many members of this association is well recognized. Here one sees that the chief insult to the lung is the tubular system itself. Variable changes of mucosa, muscularis, cartilage, and elastic membrane are described, ranging from increase in amount to decrease to absence of various portions. There is only little change in the lung parenchyma beyond collapse of alveoli, and these changes are in the contiguous areas of involved bronchi. The dilated tubular structure is seen to extend to within the margin of the pleura of the lung. Despite all of these changes in the tubular system, the pulmonary arteries have a normal appearance—they are thin walled and show no reaction.

These, then, represent the points of comparison that are being made between tuberculous and nontuberculous bronchiectasis: (1) location in the lung; (2) changes during inspiration and expiration; (3) difference in extent of tubular change as seen in the bronchogram; and (4) difference between the pulmonary arterial system in the two diseases, being greatly obliterated in tuberculosis and intact in the nontuberculous disease. Recently it has been possible to find correlation of these observations made from the injected specimens when studying patients. That is, the bronchogram observations can be made from films taken when this examination is done on the living patient and, though only a very few patients have been studied, there is a suggestion that angiocardiography with visualization of the pulmonary artery the arterial system gives similar findings. In chronic pulmonary tuberculosis the angiocardiogram reveals a paucity of arterial pattern in the involved lung area. In bronchiectasis, nontuberculous, a relatively good pulmonary arterial pattern has been seen.

The question of congenital or developmental nature of nontuberculous bronchiectasis has, to the present, not been mentioned, and is included here only to suggest a means of studying this problem. In a cystic lobe which, when removed, had classical markings of being a developmental defect, the pulmonary artery was injected, and it was noted that while the arterial pattern was present it was much smaller in caliber size, there were many fewer branches, and the entire picture was in contrast to a normal lung and to a bronchiectatic lung. In another case of what appeared to be cystic disease of the lung by bronchography, often called cystic bronchiectasis, a pulmonary angiogram showed a marked decrease in pattern. These two cases suggest that there may be a possibility of distinguishing congenital or developmental defects from acquired changes by studying the pulmonary arterial system both by angiography and direct injection of the specimen. It would seem that as an economy in nature and on teleological grounds the pulmonary arterial system might not be expected to develop normally if the need for it were not at hand as in absence of or failure of development of the lung.

FUNCTION TESTS IN PULMONARY SURGERY

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Stockholm, Sweden

GUIDED by accumulated experience, we are able to perform major pulmonary operations on a yearly increasing number of patients. These interventions more or less inevitably reduce the respiratory area of the lungs. It is very often possible to predict, without the use of complicated tests, and with a fair degree of accuracy, how much surgery may safely be contemplated. But most thoracic surgeons have certainly had the distressing experience of producing respiratory cripples from operations where the reduction of functioning lung tissue was unexpectedly excessive for the patient. On the other hand, there are patients who are at present denied the surgery from which they might derive great benefit. Could the functional prognosis in these "borderline cases" be assessed by accurate tests of respiratory and cardiac capacity, operative treatment might be successfully undertaken.

We maintain that, in order to help patients of this latter category, and to provide a more accurate basis for evaluating surgical risks, every specialized chest unit should include a well-organized laboratory for cardio-respiratory investigations. The head of the laboratory should be a "chest-minded" physiologist with his own technical staff at his disposal. Much valuable work has been done where such laboratories are already in existence, particularly at the Bellevue Hospital, New York, by Cournand and Richards and their associates.

Respiratory function is a very complicated process involving heart, lungs, blood, and tissues; tests of function fall into two principal categories: (1) those which include all the participating factors, e.g., exercise tests; (2) those in which an attempt is made to evaluate, as far as possible, the elementary functions. Exercise tests are widely used, and if satisfactory results are thereby obtained, further analysis of the elementary components may be unnecessary. But in the event of demonstrated insufficiency, i.e., if the patient is dyspneic on exertion, the elementary functions must be carefully investigated for possible causes. Dyspnea in many borderline cases does not necessarily contraindicate operation. In such patients surgery need not increase, and may even relieve dyspnea. But if, for example, pulmonary emphysema is the causative factor in respiratory insufficiency, operation is often contraindicated, or, if attempted, must be carried out with extreme caution.

FORMS OF PULMONARY INSUFFICIENCY

Dyspnea in chest patients is almost invariably of cardiac or pulmonary origin. Heart disease must, of course, be excluded by routine methods of investi-

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gation. Remaining then are the patients whose dyspnea is attributable to pulmonary anomalies.

Only ten years ago little had been done about the problem that these cases presented. During recent years, however, much new scientific research has been carried out in this field, and it is now possible to demonstrate the direct cause of dyspnea in most cases. It has been shown that most forms of pulmonary dyspnea have their origin in well-defined pathologic changes in one or more of the following physiological entities: (1) the ventilation system, including the air passages and the movements of the diaphragm and chest wall; (2) the pulmonary parenchyma, including both quantitative and qualitative changes in the respiratory membrane of the alveoli; (3) the pulmonary circulation. Consequently, the causes of pulmonary dyspnea may be grouped as follows: (1) *Ventilation insufficiency*, which may be produced by obstruction of any kind in the air passages; e.g., tuberculous strictures, tumors, enlarged bronchial glands, etc., or by impairment of costal and diaphragmatic mobility. Overlapping from this group into the next is enlargement of respiratory dead space by bronchiectasis, pulmonary emphysema, etc., which often produce severe dyspnea. (2) *Parenchymatous insufficiency*, which occurs when the size of the functioning area has been reduced: (a) quantitatively, by disease, collapse procedures, or extirpation of lung tissue; (b) qualitatively, by impaired diffusion of gases through the alveolar membrane due to toxic agents or stasis. (3) *Insufficiency of the pulmonary circulation*, which may be brought about by: (a) persistent circulation through unventilated parts of the lung, so that unoxygenated blood is mixed with arterial blood; (b) arteriovenous aneurysm, (shunt); (c) obstruction of the pulmonary artery by emboli or obliterative arteritis.

This classification is, of course, rough, and in many respects incomplete, but it is useful in practical work where it is helpful to have a working hypothesis. Naturally the categories frequently overlap, as many conditions present combined forms of insufficiency.

TESTS OF PULMONARY FUNCTION

Theoretically, pulmonary function should be investigated with exercise tests both in its entirety and with reference to its various components. In practice, however, a few procedures, which we will now discuss, and which may be carried out with the patient at rest, have proved adequate. As certain of the functions investigated, such as residual air and maximal ventilation capacity have a pronounced correlation to the degree of dyspnea, the need for exercise tests has not been greatly felt. But we feel that exercise tests are nevertheless advisable, so as to be able to form an accurate assessment of the patient's limits as regards ventilation and oxygen uptake.

RADIOLOGICAL INVESTIGATION

Fluoroscopic and radiological investigation of the costal and diaphragmatic movements is of great value. Impairment of diaphragmatic mobility by intrapleural adhesions has an especially deleterious effect on ventilatory function; it is without doubt the commonest cause of ventilation insufficiency dyspnea. A rough estimate of ventilation capacity on either side can be made by fluoroscopic study during forced breathing. We have found that registration of the maximal diaphragmatic movements by double exposure on the same film is very useful. This can be done without any special apparatus, but we prefer to use a lead grid with vertical lead bars, each ½ cm. in breadth and ½ cm. apart (Birath and Källqvist). Exposure is made at maximal inspiration; then, with the patient's position constant, the grid is shifted ½ cm. to one side and maximal expiration is registered on the hitherto unexposed strips of film. By thus covering the already exposed film strips, actual double exposure is avoided. Registration of the diaphragmatic movements in this simple way does not restrict knowledge of the ventilation potentialities to the observer behind the fluoroscopy screen. To be complete, the film must clearly show the posterior costophrenic sulcus. Lateral double-exposure films, with the patient in slightly oblique position should, therefore, also be taken. Not only can significant pleural adhesions be estimated qualitatively in this way. Impaired diaphragmatic function in emphysema and reduced costal movements in pulmonary retraction and pleural thickening can also be assessed.

SPIROMETRIC INVESTIGATION

The *total lung volume and its subdivisions* are also estimated. We wish to stress that measurement of the vital capacity alone is inadequate and may even be misleading. In emphysema, for instance, the vital capacity is often very good despite impaired lung function. Yet many of these patients do not tolerate the usual pulmonary operations (see Fig. 1). This is easily understood when we consider that vital capacity refers only to the function of the pulmonary bellows, but gives no information about the circulation or exchange of gases in the lungs.

If the total lung volume is estimated in such a patient, it is found to be surprisingly great, but the residual capacity, i.e., the amount of air remaining in the lungs after a maximal expiratory effort, is increased. Residual capacity (or residual air), normally constitutes about 25 per cent of the total lung volume, but in emphysema it is increased to 30, 40, or even 50 per cent. The increase applies also to equilibrium capacity (functional residual air, mid-capacity), i.e., the amount of air remaining in the lungs after a *normal* expiration. This comprises about 50 per cent of the total lung volume in a healthy subject, but may be as much as 60 to 70 per cent in pulmonary emphysema (Fig. 1).

Contrary to what appears to be the general opinion, emphysema is not the only cause of disproportionate increase in these values. In serial investigations of patients with various pulmonary diseases (Birath), it was found that relative increase of residual and functional residual air was present in patients with dyspnea on slight exertion or even at rest. This dyspnea could also be caused by other ventilation-impairing conditions, e.g., bronchostenosis, bronchiectasis, indurated or infiltrated parenchyma, and extensive pleural adhesions (see Fig. 2).

Previously, Hurtado and associates had stated that the degree of respiratory insufficiency in pulmonary emphysema is directly proportional to the percentage of residual air: But this dictum seems also to be applicable to other types of pulmonary dyspnea.

It was quite evident in one of the author's investigations (Birath), that this increase in the percentage of residual air was not bound to a diminished total capacity. In patients with bilateral pneumothorax, the total capacity could be reduced considerably without any significant change in the relationships between the subdivisions of the pulmonary volumina, as long as the lungs were adequately ventilated. On the other hand, patients with pronounced ventilation distress and marked dyspneic tendencies as a result of post-pleuritic pulmonary retraction showed marked disparity in these relationships; residual air was sometimes as much as 50 per cent and functional residual air, 70 per cent.

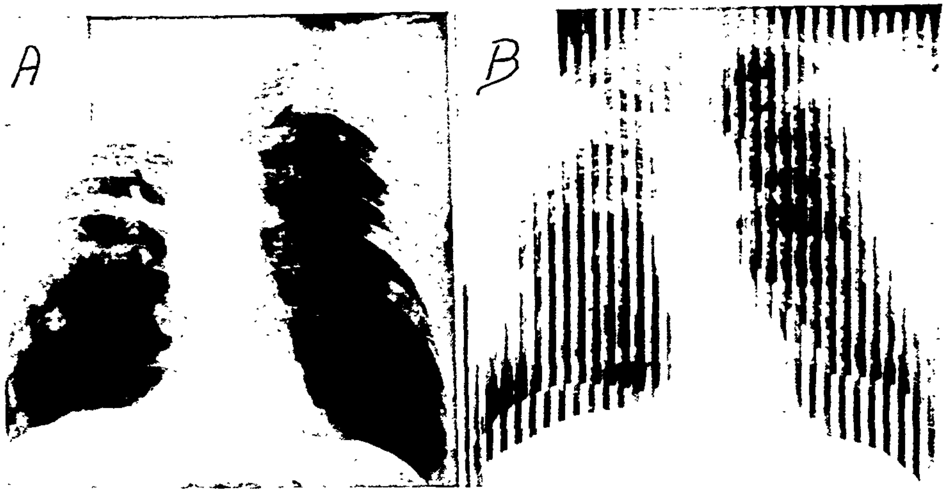

Fig. 1.—Emphysema. Unfit for operation.

A. K., a 55-year-old man. Bilateral pulmonary tuberculosis with bronchiectasis and right cavitation was diagnosed in 1949 (*A*). Double exposure on maximal inspiration and expiration shows the reduced movements and flattened contour of the diaphragm (*B*). He is dyspneic on exertion. Thoracoplasty is ruled out from a functional point of view.

SPIROMETRIC INVESTIGATION

	NORMAL VALUES (calculated)	ACTUAL VALUES
Absolute values (in liters, 37° C. saturated)		
equilibrium capacity	3.18	5.41
vital capacity	5.1	4.16
residual capacity	1.52	3.87
total capacity	6.62	8.03
Relative values (per cent of total capacity)		
equilibrium capacity	48.0	67.4
residual capacity	23.0	48.2
Maximum breathing capacity (liters per minute, 37° C. saturated)	109	31

As the fraction of special interest, residual air is fairly consistently increased in dyspnea-producing conditions, its determination gives valuable information of the functional condition of the lungs.

In our view, it is clear that indications for surgical interventions should not be bound to fixed values of the vital or total capacities. Estimation should rather be based on the percentage values of residual and functional residual air. Good vital capacity with increased residual capacity indicates inadequate rather than efficient function (see Fig. 1). Low vital capacity but normal percentage of residual air often permits pulmonary surgery (see Fig. 3).

The determination of the *"maximum breathing capacity"* according to Hermansen is also of decided value. This test demands perfect cooperation between investigator and patient, but an intelligent, willing subject can help us to obtain useful knowledge of his maximum ventilation capacity. The patient is instructed to breathe as deeply and as rapidly as possible for fifteen seconds; this is repeated at intervals until consistent values are obtained. In healthy males the range in liters per minute is 125 to 150, and in normal females 80 to 110. Cournand and Richards have found that the immediate postoperative period may be stormy, and that permanent disability in the form of dyspnea on minimal exertion or even at rest may result if the preoperative maximum breathing capacity does not exceed 30 to 40 liters per minute.

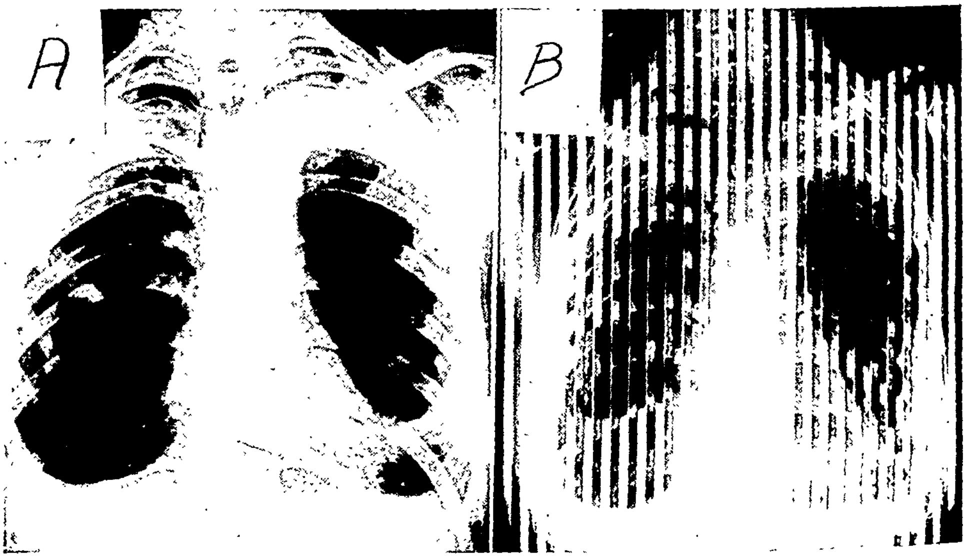

Fig. 2.—Ventilation insufficiency after bilateral pneumothorax.

S. E., a 38-year-old woman. Onset of tuberculosis (left infiltration) in 1946. Left pneumothorax was induced in July of that year. Right pneumothorax was induced in 1947 to control spread of the disease. Both pneumothoraces were discontinued in February, 1950 (*A*). Double exposure film on maximal inspiration and expiration (*B*) reveals moderate diaphragmatic insufficiency due to impairment of mobility by pleural adhesions. The patient is dyspneic on exertion.

SPIROMETRIC INVESTIGATION

	NORMAL VALUES (calculated)	ACTUAL VALUES
Absolute values (in liters, 37° C. saturated)		
equilibrium capacity	2.24	1.45
vital capacity	3.60	1.48
residual capacity	1.27	0.92
total capacity	4.87	2.40
Relative values (per cent of total capacity)		
equilibrium capacity	46.0	60.4
residual capacity	26.0	38.3
Maximum breathing capacity (liters per minute, 37° C. saturated)	87	41

BRONCHOSPIROMETRY

The foregoing tests refer to both lungs working in unison, but often do not suffice for a thorough evaluation of pulmonary function. Let us consider a

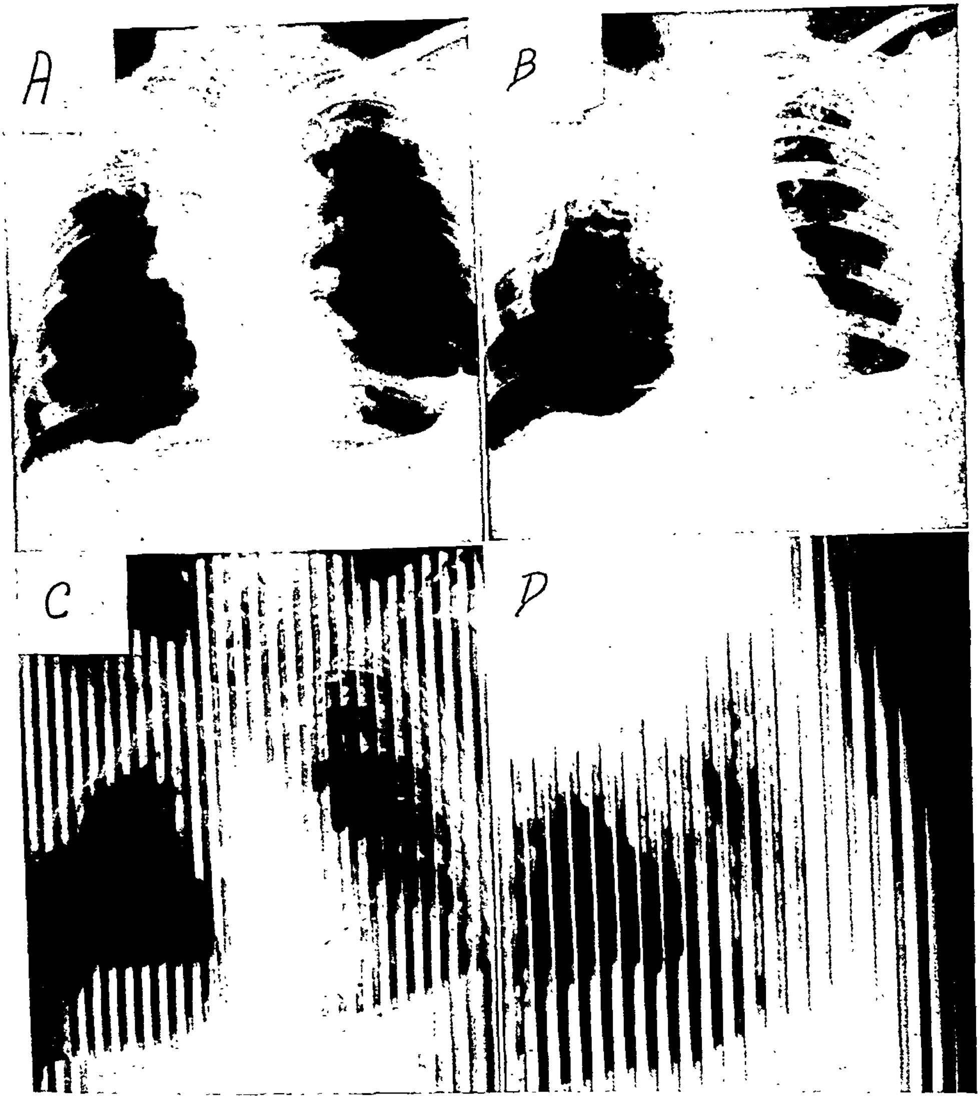

Fig. 3.—Ventilation insufficiency, fit for operation.

H. H., a 41-year-old man. Bilateral cavitary disease was discovered in 1934. Artificial pneumothorax was maintained from 1935 to 1939 on the right and from 1936 to 1942 on the left. The left pneumothorax was reestablished in 1943. Subsequently, there was bilateral impairment of diaphragmatic mobility with moderate ventilation insufficiency. The cavity in the right lung recurred and slowly increased in size. After re-expansion of the left lung and preliminary P.A.S. and streptomycin medication (*A*), two-stage thoracoplasty with extensive apicolysis was performed in August and September, 1949. Extrafascial pneumothorax was maintained in the apicolysis space for three months. The patient is now managing his iron-plating works and has no appreciable dyspnea on moderate exertion (*B*). Double exposure on maximal inspiration and expiration with grid (Birath & Källqvist) shows the movements of the diaphragm (*C, D*).

Spirometric Investigation

	NORMAL VALUES (calculated)	ACTUAL VALUES	
		BEFORE OPERATION	AFTER OPERATION
Absolute values (in liters, 37° C. saturated)			
equilibrium capacity	3.18	1.44	1.53
vital capacity	5.1	2.16	1.87
residual capacity	1.52	0.85	1.05
total capacity	6.62	3.01	2.92
Relative values (per cent of total capacity)			
equilibrium capacity	48.0	47.8	52.4
residual capacity	23.0	28.3	36.0
Maximum breathing capacity (liters per minute, 37° C. saturated)	121	44	35

patient with very limited respiratory reserves: if the pulmonary disease is unilateral, even if the lung appears to be totally destroyed by, e.g., a tuberculous process, or by neoplasmic occlusion of the main bronchus, the decision is not difficult. Total thoracoplasty or pneumonectomy will produce only temporary functional disturbance, with a subsequent return to approximately the preoperative level. But if pulmonary and/or pleural anomalies are present bilaterally, it may be impossible to decide by the above tests which lung has the better function. In such a patient, the function of each lung must be investigated separately (see Fig. 4). Should it be found that the lung requiring surgical treatment has the better function, we must act very cautiously or omit operation. But if the reverse is true, the decision is easily made, and we may proceed with confidence.

The main indication for bronchospirometry, therefore, is limitation of function by bilateral disease, most commonly tuberculosis. With some experience, it is not impossible to make a rough estimate of the function of each lung in relation to the total. Radiographs, fluoroscopic examination, double exposure in maximal inspiration and expiration give valuable help. But as even very experienced investigators may make mistakes in such patients, we are convinced that it is wiser not to evaluate roughly, but to investigate fully the pulmonary function.

We use the Björkman double spirometer, which consists of two Knipping spirometers fused into one instrument, with a common Kymographion. There are no valves in these spirometers, but the circulation of gases is ensured by means of motor-driven pumps. The flexible, double-lumen catheter used is of a new construction, devised by Carlens, our bronchoscopist. The lumen is wider than that of catheters previously in use. The catheter is introduced without the aid of fluoroscopy, and is provided with a small rubber hook that is automatically engaged by the carina. That this catheter permits the patient to breathe with very little resistance is amply proved by the fact that vital capacity is approximately the same with the catheter in situ as without it.

In judging the results of bronchospirometry, it is necessary to bear in mind that the right lung is larger and consequently has greater ventilation and oxygen consumption than the left. The ratio is approximately 53 to 47. It is interesting to note, in cases of unilateral disease, that oxygen uptake on the affected side is not necessarily directly proportional to vital capacity and ventilation in that lung. Oxygen uptake is often less than expected.

We consider oxygen the most important component to be determined, as carbon dioxide diffusion takes place much more easily, and is consistent in relation to ventilation. But Björkman and Carlens have recently shown—in investigations comparing oxygen consumption during exercise with a high oxygen content in the spirometers, and with atmospheric air—that the oxygen uptake of a diseased lung may be lower in atmospheric air than in oxygen. It is therefore probable that we must, in future, provide oxygen to maintain a stable composition of air in the spirometers.

Complications arising from bronchospirometry are rare and would appear to be almost completely avoidable, provided that the patient has little or no

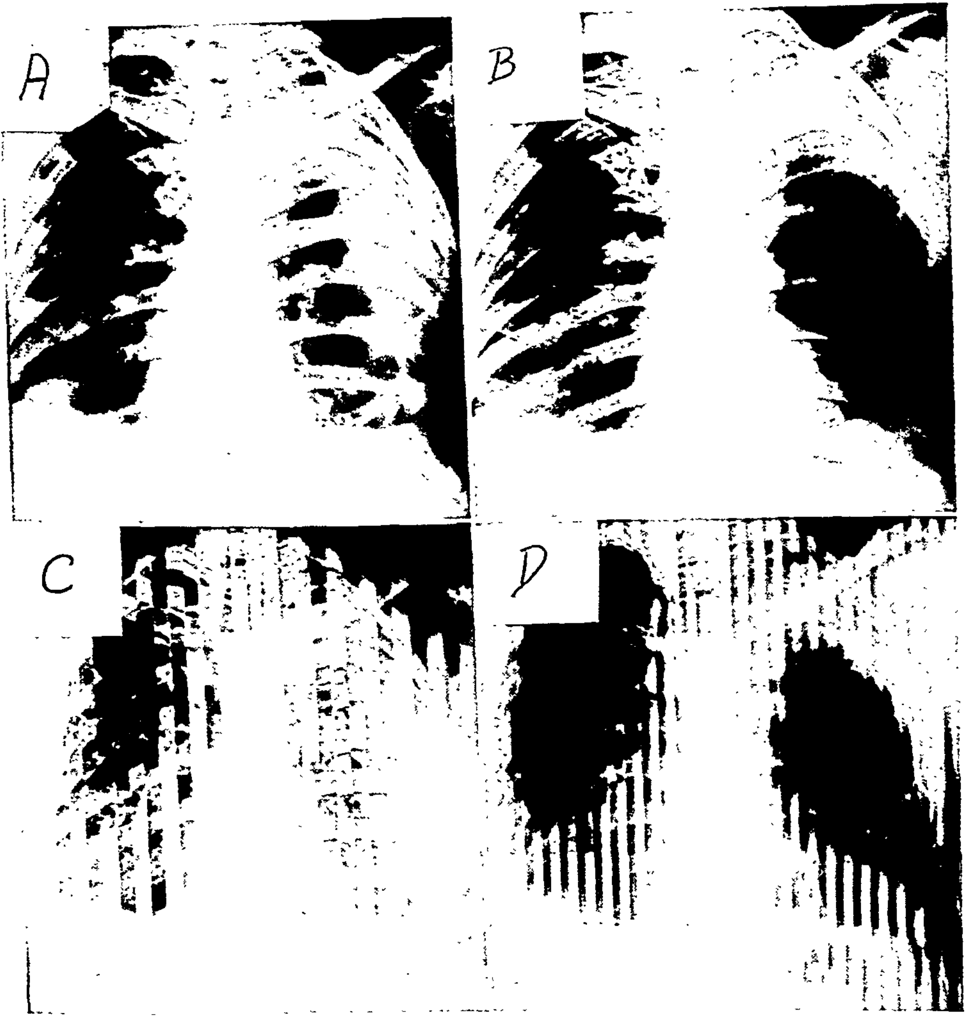

Fig. 4.—Ventilation insufficiency, improved after decortication.

E. K., a 41-year-old man, in 1943 was found to have bilateral apical infiltration with left cavity. Left artificial pneumothorax was attempted but was abandoned because of adhesions. Left extrapleural pneumothorax was performed in 1944. A severe postoperative hemorrhage caused reformation of the apical adhesions. By the end of 1945, further refills were impossible. The lung was encapsulated in grossly thickened pleura (*A*), and the patient was dyspneic in the extreme. Double exposure on maximal inspiration and expiration shows very limited movements of the diaphragm and chest wall (*C*). After preliminary P.A.S. medication, decortication of the left lung was performed in February, 1950, with great amelioration of subjective distress (*B*) and improved ventilation movements (*D*).

SPIROMETRIC INVESTIGATION

	NORMAL VALUES (calculated)	ACTUAL VALUES	
		BEFORE OPERATION	AFTER OPERATION
Absolute values (in liters, 37° C. saturated)			
equilibrium capacity	3.18	1.18	1.83
vital capacity	5.1	1.31	1.93
residual capacity	1.52	0.83	1.22
total capacity	6.62	2.14	3.15
Relative values (per cent of total capacity)			
equilibrium capacity	48.0	55.1	58.1
residual capacity	23.0	38.8	38.7
Maximum breathing capacity (liters per minute, 37° C. saturated)	120	38	56

BRONCHOSPIROMETRY

	BEFORE OPERATION					AFTER OPERATION				
	R	L	R+L	%R	%L	R	L	R+L	%R	%L
O_2 consumption per minute	0.285	0.033	0.318	89.6	10.4	0.244	0.081	0.325	75.1	24.9
Ventilation per minute	11.00	2.60	13.60	80.9	19.1	6.732	2.904	9.636	69.9	30.1

sputum and is in good general condition. The single case we have observed of acute spread of the tuberculous process to the basal parts of the lung was rapidly controlled by a course of combined para-aminosalicylic acid and streptomycin.

HEART CATHETERIZATION

The more pulmonary function is scientifically explored, the more obvious becomes its integration with the right heart. So intimate are the two that they seem to constitute a functional unit. In healthy persons, increased blood flow through the lungs appears to be ensured by the opening of a large number of reserve capillaries: these are closed in the resting state, but during exercise the active area of the vascular bed may increase ten to twenty times without significant rise in blood pressure. In diseases affecting the pulmonary blood-vessels, e.g., emphysema and tuberculosis, the area of the pulmonary vascular bed is reduced and the pressure in the minor circulation consequently increased. If the strain on the right heart becomes excessive, right cardiac insufficiency develops, with its characteristic dyspneic attacks, often in the almost complete absence of other symptoms of cardiac failure.

In order to avoid this right cardiac involvement, we have commenced preoperative registration of the pressure in the right pulmonary artery; catheterization is performed with the technique in common use. The fullest information seems to be obtained if some form of graded exercise is undertaken during the test so that the pressure reaction may be observed. This method is carried out by Hansson at Sabbatsbergs Hospital. It is too early as yet to estimate the significance of the procedure and to give normal values, but theoretically it appears to be most valuable.

Now that chemotherapy is so extensively used, more and more tuberculous patients come into the category of "arrested disease," even advanced cases with persistent cavities. Many of these patients have had tuberculosis for many years, and have greatly reduced pulmonary parenchyma. There is also considerable cardiac strain, and it may be difficult to decide if more may be added. In these patients, as well as in emphysematous subjects, the determination of the circulatory reaction during exercise must be of the utmost value.

SUMMARY

To obtain widened indications for pulmonary surgery in patients who, because of impaired function, would otherwise be deemed unfit for operation, pulmonary function should be evaluated by analysis.

The writers differentiate between: (1) *ventilation insufficiency,* which arises from bronchial anomalies or reduced costal and diaphragmatic mobility; (2) *parenchymatous insufficiency,* quantitative (loss of parenchyma), or qualitative (impaired diffusion of gases); (3) *insufficiency of the pulmonary circulation* (blockage or shunt).

The routine methods employed by the writers are: (a) *Radiological examination,* with registration of the movements of the diaphragm and thoracic cage. (b) *Spirometric investigation,* which includes the total lung volume and its

subdivisions and the maximal ventilation capacity. Special importance is attached to the relationship between the residual and total capacities and between the equilibrium and total capacities. Disparity in these relationships often heightens with increasing dyspnea. (c) *Bronchospirometry,* which is performed when other investigations show significant impairment of *total* lung function. (d) In certain cases, *catheterization of the pulmonary artery,* to determine the pressure at rest and during exercise.

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NOCARDIOSIS

Report of a Single Case With Recovery

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IN REVIEWING the literature, there are strikingly few cases of Nocardiosis reported, and the diagnosis in the great majority of cases was made either at autopsy or in the late stages of the illness. With the exception of a single case of Madura foot cured by amputation, only an occasional recovery has been reported, and those have occurred since the advent of sulfonamides.

Nocard[1] described an acid-fast sporothrix in 1888 as the cause of bovine farcy. In 1890, Eppinger[2] isolated an aerobic gram-positive acid-fast actinomyces from a fatal case of multiple brain abscesses and meningitis in man, describing this organism under the name *Cladothrix asteroides.* Since then the species of Actinomycetes having acid-fast properties have been described under various names. Finally the aerobic acid-fast Actinomycetales not forming condia were placed in the genus Nocardia. Many species of acid-fast Nocardia have been described, but in 1939 Umbreit[3] reduced these to the status of strains or variants of *Nocardia asteroides.* The genera Nocardia and Mycobacterium are closely related morphologically and culturally, but Drake and Henrici[4] concluded (1943) in their paper that the allergy induced against *N. asteroides* was specific, and that there were no cross reactions to tuberculin.

The first extensive review of the literature on this subject was in 1921, when Henrici and Gardner[5] collected twenty-six cases of acid-fast actinomyces, adding a single case of their own. The latter was a patient suffering from what appeared to be a pulmonary infection, who was treated with antigen, but the outcome was uncertain. Of the group of twenty-six cases there was only one recovery, and this was a patient with Madura foot who recovered following amputation. Since then single fatal cases have been reported by Goldsworthy[6] in 1937, and Kessel and Goolden[7] in 1938.

In 1944, Benbow, Smith, and Grimson[8] of this institution reviewed the literature and reported two new cases of *N. asteroides* with pulmonary and chest wall infections. Both of these patients were treated successfully with bed rest, surgical drainage, vitamins, sulfonamides, and iodides. These were the first patients known to have recovered, except for the previously mentioned patient with Madura foot.

In 1945, Binford and Lane[9] reported a fatal case, the diagnosis being made at autopsy. In 1946, Kirby and McNaught[10] added two fatal cases and also reviewed the literature, stating that of the thirty-two known cases of *N. aster-*

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oides, twenty-eight were dead at the time the report was made. Their two cases brought the total to thirty-four, only three of whom were known for certain to have survived, and one of the latter was the case of Madura foot.

In 1946, there were also reports of two single cases with recovery, one by Shaw, Holt, and Ray,[11] and the other by Calero.[12] The case reported by the former received penicillin, thymol, potassium iodide, sulfadiazine, and surgical drainage of an empyema cavity, while the case reported by Calero apparently received sulfonamides and penicillin in the course of treatment.

In 1948, Glover, Harrell, Heilman, and Pfuetze[13] reviewed the literature and added a case of a 26-year-old white male physician with an *N. asteroides* infection simulating pulmonary tuberculosis. Their patient was successfully treated with intensive sulfadiazine therapy, whereas penicillin and streptomycin failed to control the infection. The authors stated that from studies carried out in their laboratories and elsewhere, sulfadiazine appeared to be the drug of choice in the treatment of Nocardiosis.

In 1949, Hager, Migliaccio, and Young[14] reviewed the literature, and reported two additional cases. One was a 16-year-old schoolgirl with a pulmonary infection and empyema who was successfully treated by surgical drainage and combined therapy with sulfadiazine, penicillin, and streptomycin. The second case was that of a 64-year-old woman with bilateral apical pulmonary lesions. *N. asteroides* was isolated from the sputum, and by studies in vitro was found to be inhibited by penicillin, aureomycin, and streptomycin, but not by sulfadiazine in levels feasible to obtain clinically. There was no follow-up on this patient. They emphasized that it was necessary to observe patients carefully for several years for signs of recurrence.

Tucker and Hirsch[15] also reviewed the literature in 1949 and added three cases of their own, two of which terminated fatally. They mentioned that geographically the distribution of the aerobic type of Actinomycosis is worldwide, that there apparently is no occupational incidence, and that the disease appears to be fairly well distributed throughout adult life. They concluded that Nocardiosis should be considered in all patients with obscure or unusual chronic infections, especially when repeated attempts to demonstrate the tubercle bacillus have failed.

CASE HISTORY

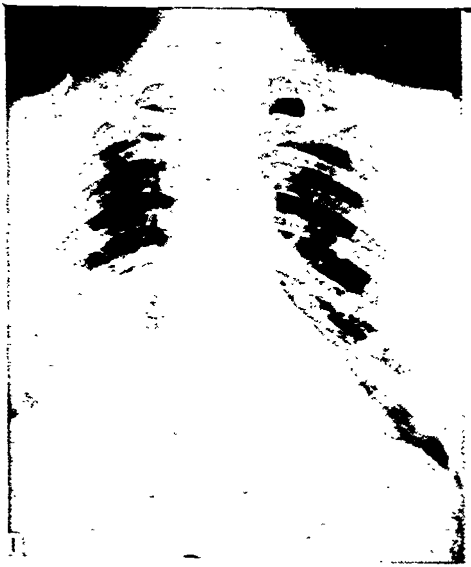
CASE 1.—A. W., C-82777. This 37-year-old white tobacco farmer was referred to Duke Hospital on March 10, 1950, for investigation of a mass in the right lung field. One month prior to admission, because of fever, cough, and right anterior chest pain, he was admitted to his local hospital with a diagnosis of virus pneumonia. Roentgenogram of the chest showed a shadow in the right lung field. He was treated with penicillin without improvement or change in the roentgenogram picture, but it was stated that during his hospital stay he became afebrile. His cough was productive of approximately 2 tablespoonfuls of blood-streaked white mucoid sputum per day. There was an associaed 20 pound weight loss during the month in spite of a fair appetite. Because of persistence of symptoms he was transferred to Duke Hospital for further investigation.

The family history was of interest in that a sister had died thirteen years previously with pulmonary tuberculosis, and there was some question as to whether the patient's mother had tuberculosis. The patient's general health had always been good, and his only recent illness had been a single attack of "malaria" in 1948. He had been obese all of his adult life, with his average weight being around 200 pounds and his weight at the time of admission being 180 pounds.

Physical Examination.—Temperature, 37° C.; pulse 80; respirations, 20; blood pressure, 148/100. The patient was a well-developed, obese, cooperative white man who did not appear ill. The examination was occasionally punctuated by a nonproductive cough. The skin was without lesions, and there was no lymphadenopathy demonstrable. Examination of the head, eyes, ears, nose, and throat was essentially normal. The trachea was in the midline, and there was no venous distention seen in the neck. The chest was barrel-shaped, but was symmetrical, and there was no respiratory lag. There was dullness to percussion in the right lower lung field anteriorly and posteriorly. Over this same area there were decreased breath sounds and diminished tactile fremitus. No râles were heard. The heart was not enlarged by percussion. The rhythm was regular, and there were no murmurs. The abdomen was protuberant, but there were no masses, tenderness, or rigidity. There was an easily reducible left inguinal hernia. The remainder of the physical examination, including rectal and neurological examinations, was essentially normal.

Accessory Clinical Findings.—Hemoglobin 15.3 Gm.; white blood cells, 20,000; platelets, adequate. Differential: segs. 76, stabs 5, juv. 4, eosinophiles 2, lymphocytes 13. Urine: pH 6.0. Specific gravity, 1.028. Protein and sugar, negative. Microscopic 0-1 white blood cell per high-powered field. No casts or bacteria. Posteroanterior stereoscopic and lateral films of the chest showed a density which seemed to be confined to the area of the right middle lobe (Figs. 1 and 2). Planigrams through this area revealed no cavities or discrete areas of calcification. Barium examination of the esophagus, stomach, and intestinal tract was normal. Cardiolipin test for syphilis was negative. Old tuberculin skin test, 1:1000 dilution, was 1 plus in 48 hours. Skin tests to blastomycin, coccidioidin, and histoplasmin were all negative. During the initial period in the hospital the patient brought up very little sputum, the latter being mucoid in nature and containing no blood. Routine cultures of the sputum grew *Neisseria sicca*, nonhemolytic *Micrococcus aureus*, a nonhemolytic Streptococcus, and *Streptococcus viridans*. Three concentrated sputum examinations were negative for acid-fast bacilli. Five separate sputum specimens cultured for tubercle bacilli were negative. One examination of the sputum for fungi was negative, and one culture for fungi was negative. On another occasion a routine culture of the sputum grew out an aerobic actinomyces, which was considered at the time to be a nonpathogen and a contaminant. Bronchoscopy revealed thick purulent secretions coming from the middle lobe orifice and narrowing of the middle and the lower lobe orifices by bronchitis. No definite tumor mass was seen. Specimens for Papanicolaou smears from the area revealed no tumor cells. Smears and cultures of the secretions for tubercle bacilli were negative. A routine culture of the secretions aspirated through the bronchoscope grew an aerobic actinomyces in eight days which was thought at the time to be a nonpathogenic contaminant.

Course in Hospital.—Throughout the fourteen-day period of observation and diagnostic survey the patient ran a low grade fever (37.5° to 38° C.) of the nonseptic type. He brought up little sputum until the fifth day after admission, at which time he was bronchoscoped. Following this, the amount of sputum increased to between 90 and 380 Gm. of mucopurulent sputum a day. During this preliminary period he was treated with intramuscular penicillin 300,000 units a day and penicillin aerosol 100,000 units three times a day. At the end of a two weeks' period no definite diagnosis could be made, so an exploratory thoracotomy was performed on March 23, 1950. At operation the pleural cavity was found to be completely free without fluid. There was complete consolidation of the entire middle lobe, and an area of involvement of the contiguous portion of the lower lobe measuring approximately 5 cm. in diameter. There was no involvement of the upper lobe. On dissecting in the major and minor fissures a thick, yellow, purulent material was encountered. It was the operator's impression that he was dealing with an inflammatory lesion rather than a neoplastic lesion, the etiology most likely being tuberculous. A right middle and lower lobectomy was then performed. The chest was closed leaving two drainage catheters in place after penicillin and streptomycin had been instilled. Postoperatively he was treated with a total of one gram of streptomycin and 300,000 units of penicillin intramuscularly a day. An equivalent amount of the two drugs was placed in the chest each day. The postoperative course was complicated by collapse of the remaining right upper lobe, but with vigorous suctioning and coughing the lung re-expanded, and the course from then until the time of discharge on the eighteenth postoperative day was uneventful. The sections of the removed lung re-



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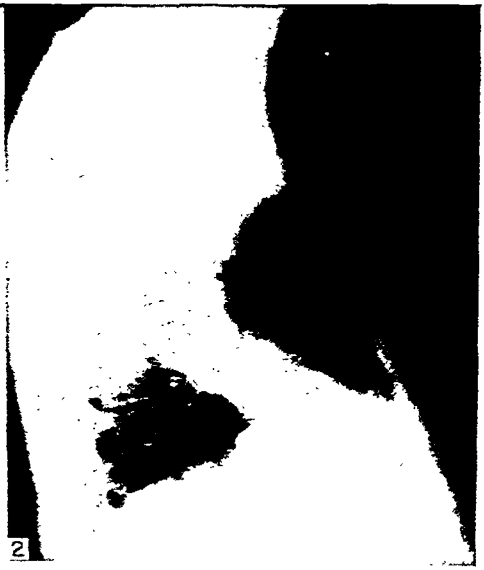

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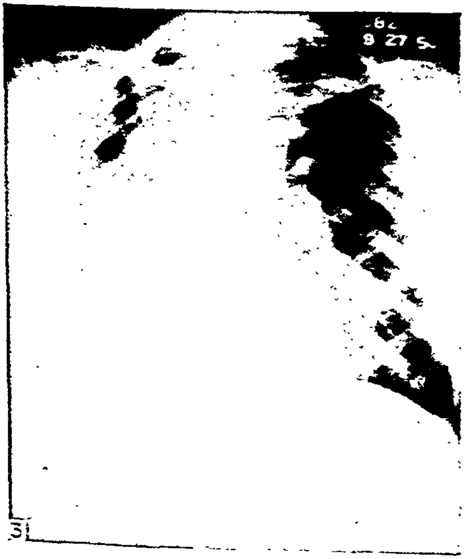

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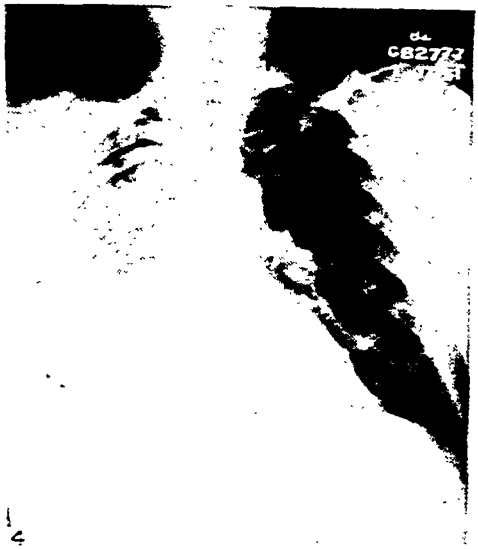

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Fig. 1.—Posteroanterior roentgenogram of the chest taken on the day of patient's first admission to the hospital, March 10, 1950.

Fig. 2.—Lateral roentgenogram of the chest, March 10, 1950.

Fig. 3.—Posteroanterior roentgenogram of the chest four months after discharge from the hospital, Sept. 27, 1950. Residual lipiodol from injection of empyema cavity can be seen in the right chest.

Fig. 4.—Posteroanterior roentgenogram of the chest eight months after discharge from the hospital, Jan. 17, 1951.

had tuberculosis. The patient's general health had always been good, and his only recent illness had been a single attack of "malaria" in 1948. He had been obese all of his adult life, with his average weight being around 200 pounds and his weight at the time of admission being 180 pounds.

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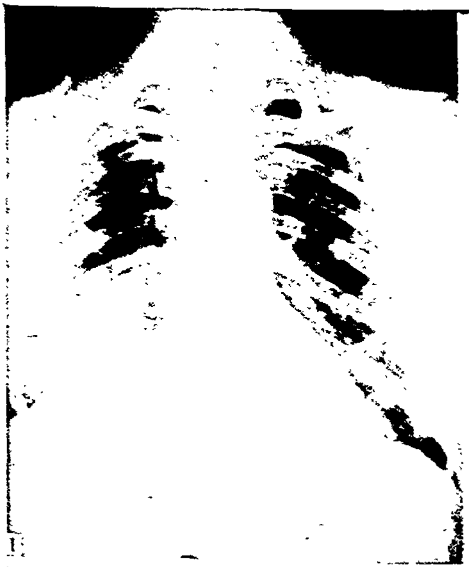

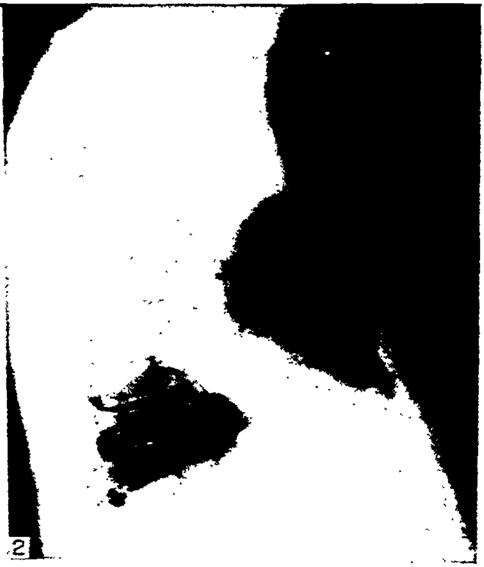

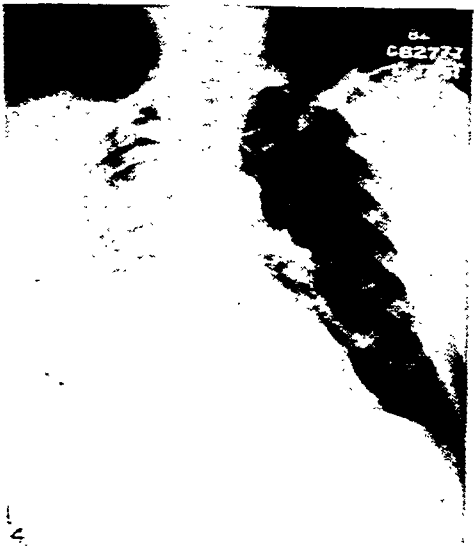

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Fig. 4.—Posteroanterior roentgenogram of the chest eight months after discharge from the hospital, Jan. 17, 1951.

vealed the middle lobe to be replaced by an indurated atelectatic parenchyma within which were noted multiple small foci of pale white areas containing a creamy-like material which grossly resembled caseation necrosis. Microscopic sections were characterized by a chronic inflammatory response consisting chiefly of lymphocytes and macrophages. In some regions the macrophages were loaded with fats. No characteristic granulomatous foci were encountered. Gram stains of the sections were not done. The lesion in the pathologist's opinion most closely resembled a chronic unresolved pneumonia in the nature of a lipoid pneumonia with multiple small abscess formations. The patient was discharged from the hospital on April 10, 1950, to be followed in the Out Patient Chest Clinic.

Second Admission: April 25, 1950. In the three-week interval during which the patient was home he did well until seventy-two hours before the second admission, when he developed an intermittent severe pain in the right chest and a cough productive of a small amount of white mucoid sputum accompanied by a mild headache and temperature elevation.

On admission the temperature was 40° C., respirations 35, pulse rate 100, blood pressure 140/90. He appeared acutely ill, and there was a slight cyanotic tinge to his lips. His skin was hot and moist. Examination of the eyes, ears, nose, and throat was as on the previous admission. The trachea was in the midline. There was a well-healed anteroposterior thoracotomy scar over the lateral aspect of the right chest. There was no redness or fluctuation in the operative scar. There was a dullness to percussion which changed to flatness from above to below over the right chest anteriorly and posteriorly. Breath sounds were absent over the right lower chest. There were a few râles at the right apex. The left chest was clear. Examination of the heart and abdomen was not remarkable. There was no calf or thigh tenderness, and the remainder of the physical examination was essentially normal.

Accessory Clinical Findings.—Hemoglobin, 10 Gm.; white blood cells 12,500. Differential: seg. 82, stabs 2, juv. 1, monocytes 3, lymphocytes 12. Urine showed no protein or sugar. Microscopic: 32 white blood cells per high-powered field, no casts or bacteria. Roentgenogram of the chest on the night of admission showed a haziness over the entire right lung field. Blood cultures revealed no growth.

During the first five days in the hospital the patient ran an extremely septic course, with temperature spikes to around 39.5° C. each day. This occurred in spite of two grams of aureomycin and 150,000 units of penicillin aerosol per day. Thoracenteses were done on several occasions without obtaining any fluid. On the fifth hospital day an abscess pocket was found in the sixth interspace posteriorly, and 15 c.c. of thick, purulent material were aspirated. On the following day the patient was taken to the operating room, and a large empyema cavity was drained. Because of the consistency of the purulent material, which was almost semisolid in nature, azochloramid irrigations to the empyema cavity were started. However, the patient continued to run a septic course with temperature elevation to above 39° C. each day for the next five days. Then the patient developed a draining sinus at the site of a previously made wound where an intercostal catheter had been located. This tract was further opened and a second empyema cavity drained.

The acid-fast cultures of the material aspirated from the posterior empyema cavity were negative. On routine culture the plate was again overrun by an aerobic actinomyces which was thought to be a contaminant, but in view of its abundance suspicions were aroused, particularly since material obtained from both of the empyema cavities at the time of drainage grew a pure culture of this fungus. Material from each of these empyema cavities was turned over to Dr. N. F. Conant of the Mycology Department who identified the organism as *Nocardia asteroides.* Several fresh preparations of the material were negative, but on Gram stain, gram-positive branching filaments could be identified. There were no other organisms obtained from either empyema cavity. Identification of the organism was made by the ninth hospital day, and the patient was started on 2 Gm. of sulfadiazine per day. Due to an error this was continued for five days, and had no effect on the patient's temperature. On the fourteenth day the mistake was discovered, and the dose was increased to 1 Gm. each of sulfamerazine and sulfadiazine every four hours, amounting to a total of 12

Gm. of sulfonamide per day. The urine was alkalinized with molar lactate solution and fluids were forced to 4000 c.c. per day. On the day following, the maximum temperature was 38.3° C., the first time since admission that it had not risen to 39° C. or above. Within four days the patient was afebrile. On this dosage of sulfonamide he gradually developed a microscopic hematuria, and the nonprotein nitrogen rose to 70 mg. per cent. For this reason the dosage of sulfonamide was cut in half, making a total of 3 Gm. each of sulfadiazine and sulfamerazine per day. Daily sulfonamide blood levels were done throughout the period of therapy, and the levels ranged from 17.5 to 25.2 mg. per cent, with an average around 20 mg. per cent. After reduction of the sulfonamide dosage the hematuria rapidly cleared, and the nonprotein nitrogen fell to normal limits. He was given several supportive blood transfusions. The drainage from the anterior empyema cavity rapidly ceased, and the drains were removed prior to discharge. The tube in the posterior cavity was withdrawn more slowly. During the last sixteen days of his hospital stay he was ambulatory and afebrile.

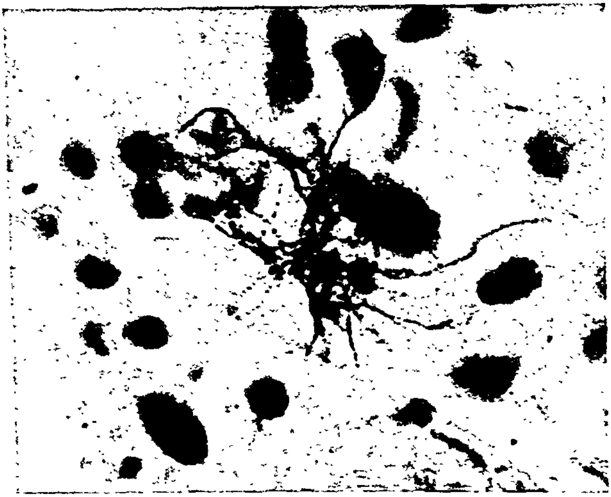

Fig. 5.—Gram-stained smear of the purulent material obtained from the empyema cavity at the time of surgical drainage. The gram-positive, delicate branching hyphae of *Nocardia asteroides* are readily seen.

He was discharged from the hospital on the twenty-sixth hospital day, May 28, 1950, to the care of his local doctor for biweekly changes of his surgical dressings, and biweekly hemoglobin, white blood cell, urine, and nonprotein nitrogen determinations. The same dosage of sulfonamide level was to be carried out for at least a three months' period of time.

Since discharge from the hospital the patient has been followed in the Surgical Out Patient Clinic at monthly intervals. Each time he has continued to show improvement. The posterior catheter was gradually removed during the month following discharge, and the wound promptly healed. A culture of the material from the sinus prior to its closure was negative for fungi. The nonprotein nitrogen and white count have been within normal limits on each occasion. The urine has shown no red cells. The sulfonamide level was 11 mg. per cent. The hemoglobin has ranged around 11 grams. The patient has gained in strength and weight. On Sept. 6, 1950, he developed a pain in the right lower quadrant with fever and elevation of his white count. He was taken to another hospital where under spinal anesthesia an appendectomy was performed. The operator stated that the appendix was acutely inflamed without evidence of chronic granulomatous process. The McBurney incision healed promptly without evidence of suppuration. Unfortunately the specimen was not saved.

The patient was kept on sulfonamide therapy from May 3, 1950, until Oct. 7, 1950, a total of five months of therapy. Since the discontinuation of drugs the patient has continued to do well and has had no complaints.

He was last seen in the Out Patient Clinic, April 12, 1951. The thoracotomy and empyema drainage incisions were well healed, and his chest film showed no evidence of activity of the old disease process (Figs. 3 and 4). He had gained approximately 80 pounds and had no complaints.

MYCOLOGY

Cultures of *Nocardia asteroides* were obtained from sputum, from secretions obtained by bronchoscopy, from lung tissue obtained at lobectomy, and from pus obtained from the posterior empyema cavity. The first isolations were thought to be contaminants because of the somewhat white chalky surface of the colonies and the slight earthy odor of the cultures which are characteristics of the aerobic soil actinomycetes, *Streptomyces sp.* When gram-stained and acid-fast stained smears of the material from the empyema cavity were examined, however, they revealed gram-positive and partially acid-fast, delicate, branching hyphae 1 micron in diameter (Fig. 5). Since such an appearance is characteristic of *Nocardia asteroides,* all cultures were re-examined and found to be consistent with this diagnosis.

Growth developed on blood agar plates at 37° C., in four to six days, as small, dry white chalky colonies with a slight earthy odor. On Sabouraud's glucose agar at room temperature (Fig. 6) the granular colonies produced an orange pigment with a thin powdery white surface. Microscopically, the culture was composed of long, delicate, branching hyphae 1 micron in diameter. When smears were made for Gram stains, the hyphae were seen to break up and appear as gram-positive bacillary elements. The fungus was also found to be partially acid-fast.

Two guinea pigs were inoculated intraperitoneally with heavy saline suspensions of colonies from Sabouraud's glucose agar slants and were sacrificed in two and three weeks, respectively. Few lesions were seen in these animals, and the organism could not be demonstrated by stained smears of various organs or peritoneal fluid. The organism was cultured, however, from a lesion in the liver of one of the guinea pigs. A third guinea pig was inoculated with a heavier suspension of the organism, and was found dead at the end of ten days. The peritoneum was studded with numerous, small, caseous nodules, and there were numerous enlarged mesenteric lymph nodes, which on sectioning were seen to contain yellowish white purulent material. Gram stained smears of the latter revealed *N. asteroides.*

PATHOLOGY

All of the sections of the involved lung were reviewed. There was a variety of inflammatory reactions, the most prominent of these being a chronic inflammatory response consisting chiefly of lymphocytes and macrophages. The overlying pleura was thickened, congested, and edematous, presenting the picture of an acute fibrinous pleurisy with many polymorphonuclear leucocytes in the subpleural connective tissues. Less conspicuous were areas of fibrosis in which there were giant cells and lymphocytes. Changes in the alveolar pattern of the lung varied from place to place, depending upon whether the predominant inflammatory response was acute or chronic. In the acutely involved area, the earliest changes seen were those of hyperemia and edema with a few polymorphonuclear leucocytes in the alveoli. The next change seen was that in which the alveoli were plugged with masses of polymorphonuclear leucocytes and small amounts of fibrin. If the process continued, the end result was the formation of numerous abscesses composed of polymorphonuclear leucocytes in all forms of preservation, fibrin, and tissue debris. Some of the abscesses communicated with bronchioles, and where this was seen the bronchiolar wall adjacent to the abscess was necrotic, the lumen being filled with polymorphonuclear leucocytes. In other areas the interlobular septae were thickened, and the alveoli were filled with edema fluid and contained many large foamy macrophages, lymphocytes, and polymorphonuclear leucocytes. Many of the alveolar walls themselves were thickened and contained scattered lymphocytes. About the previously mentioned areas of abscess formation there was a limiting zone of dense fibrous tissue containing chronic inflammatory cells. Occasionally, evidence of an organizing pneumonia could be seen.

The offending organism could not be seen in ordinary hematoxylin and eosin preparations. Bacterial stains made on the tissue sections after the diagnosis had been established revealed the presence of delicate branching mycelia 1 micron in diameter which were gram-positive, but were not easily detected by the unexpectant eye (Fig. 7). Ziehl-Neelson stains revealed the organism to be weakly acid-fast with a tendency to take up the methylene blue counterstain in the tissue sections.

The pathologic findings differed from tuberculosis in that there were no epithelioid tubercles or caseation.

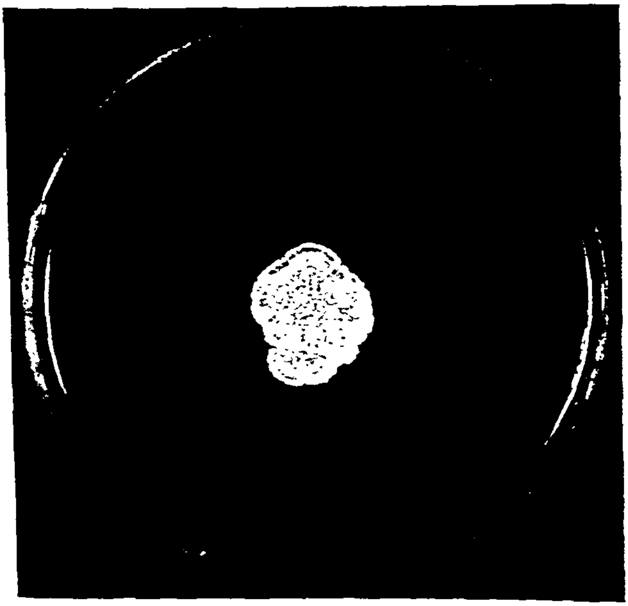

Fig. 6.—Culture of *Nocardia asteroides* grown on Sabouraud's glucose agar at room temperature for five weeks.

TREATMENT

As previously mentioned, of the reported cases of Nocardiosis definitely known to have survived, with the exception of the case of Madura foot, all received sulfonamide therapy in one form or another in conjunction with other forms of treatment. Hager and associates[14] reported a case of a 64-year-old woman with bilateral apical pulmonary lesions and a chronic productive cough. *N. asteroides* was isolated from the sputum as the predominating organism on several occasions. In vitro sensitivity tests revealed this particular strain to be inhibited by penicillin, aureomycin, and streptomycin, but not by sulfadiazine. Apparently no further tests were made with other sulfonamides. There is no follow-up on this patient, and it is merely stated that she was afebrile during a short period of observation.

Strauss and associates,[16] in their report on the chemotherapy of Actinomycosis and Nocardiosis, did in vitro studies on ten different strains of *Nocardia asteroides* with fourteen chemotherapeutic agents. The greatest inhibition was seen with aureomycin, chloramphenicol, terramycin, Myvizone, and N,N-bis (thiocarbamyl) hydrazine. Sodium sulfadiazine was only moderately effective in vitro against all of the strains tested. On the other hand, when animal protection tests were used, sodium sulfadiazine afforded almost 100 per cent protection, while the above-mentioned chemotherapeutic agents gave only partial protection.

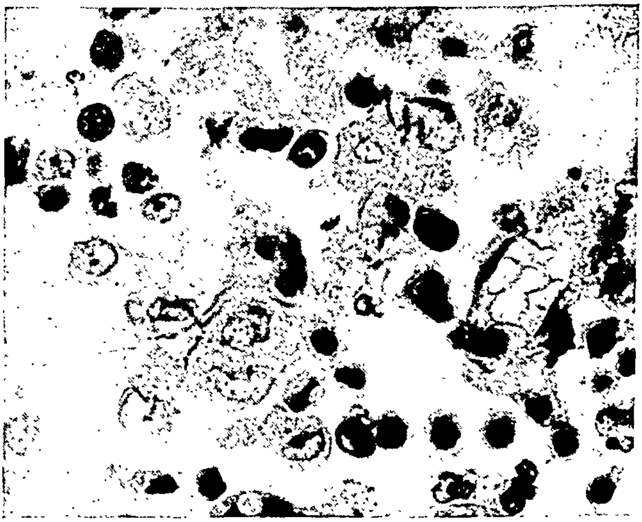

Fig. 7.—Gram stain of a tissue section from the involved lung. Short gram-positive hyphae can be seen.

In our case in vitro sensitivity tests were not done. Following large doses of sulfonamides the patient became afebrile in four days. Sulfadiazine and sulfamerazine were used jointly rather than one or the other in hopes of lessening the chance of renal complications in view of the large dosage of sulfonamides deemed adequate for treatment. We strove to maintain blood levels ranging around 20 mg. per cent while the patient was in the hospital and felt that it was quite important that the patient be kept on sulfonamide therapy for several months. Needless to say, patients under this vigorous regimen of sulfonamide therapy must be watched closely for renal complications and leucopenia.

During this patient's hospitalization he received large doses of parenteral penicillin, aerosol penicillin, and streptomycin and aureomycin at one time or another, none of which had a specific effect on the temperature curve. He also received accessory vitamins and iron. Surgical drainage undoubtedly was an essential part of his treatment.

Of the forty-two cases of Nocardiosis reported in the literature through 1949, excluding the case of Madura foot, only seven can be considered clear-cut examples of recovery. This is the third case to have recovered at this institution[8] and brings the total to eight recoveries of forty-three reported cases.

CONCLUSIONS

1. The literature is reviewed. There are strikingly few cases of Nocardiosis reported. This is most likely due to failure of recognition of many cases.

2. The case of a 37-year-old white man who was explored for a possible pulmonary neoplasm and subsequently found to have Nocardiosis is reported. The patient survived following drainage of an empyema cavity and intensive sulfonamide therapy, and is living and well one year later.

3. Nocardiosis is caused by *Nocardia asteroides*, an aerobic, gram-positive, partially acid-fast actinomyces. The mycology of this case is discussed.

4. Pathologically one sees both an acute and chronic inflammatory reaction which differs from tuberculosis by the absence of epithelioid tubercles and caseation.

5. Of forty-three cases of Nocardiosis reported in the literature (including this case) only eight can be considered clear-cut examples of recovery. Three of the latter have been reported from this institution. Of those recovering all received sulfonamide therapy in one form or another. It is essential that these patients receive adequate dosage of sulfonamides and apparently be maintained on therapy for four to six months.

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PULMONARY RESECTION FOR HISTOPLASMOSIS

A CASE REPORT

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GREAT interest has been manifested in histoplasmosis in recent years. The discovery of a useful skin test for this infection has revealed its widespread prevalence in the lower Mississippi Valley Basin. The vast majority of persons with a positive skin test are apparently normal with only some pulmonary calcifications remaining as evidence of the obsolete infection. At the other extreme there have been described a small number of highly fatal cases of disseminated histoplasma infection. Between these two extremes there have been noted a small number of patients, usually children, with early infiltrations due to histoplasma. These have usually cleared without evidence of serious illness. The case to be described below does not fit into any of the above categories. It is, therefore, felt to be of interest, particularly since it may easily be confused with pulmonary tuberculosis. This is also one of the earliest diagnosed cases to be treated by pulmonary resection. No review of the literature will be given here since this has been adequately covered in recent publications.[1]

CASE REPORT

Mrs. M. T., born in 1914, had resided most of her life on farms in Ohio and Illinois. Her past history was noncontributory. The present illness apparently began in April, 1948, with a sore throat which persisted for about two months. This was followed by rhinitis and sinusitis for two weeks. These subsided and were promptly followed by evidence of lower respiratory infection with cough. In July, 1948, she improved but continued to feel fatigued. By September the fatigue increased, she had more cough, and complained of pain in the chest. There was very little sputum, no hemoptysis, no night sweats. Her private physician referred her to the Livingston County Tuberculosis Sanitarium. Physical examination (Oct. 11, 1948) was essentially normal, except for fever to 100° F. She was ambulatory and did not appear acutely ill. No definitely abnormal findings were noted in the examination of the chest. Roentgenograms on admission showed abnormal densities above the right interlobar fissure and lateral portions of the right lung, extending upward to the clavicular level. There was some rarification noted suggestive of a motheaten type of cavitation. Mantoux tests with tuberculin PPD first strength and second strength were negative. Repeated search of the sputum for tubercle bacilli was negative. Gastric lavage specimen was negative for tubercle bacilli on culture and guinea pig inoculation. Sedimentation rate was 26 mm. Weight was 115 pounds. Blood count was within normal limits. Undulant fever and Kahn test were negative. The patient remained in the sanitarium until Jan. 24, 1949, with considerable symptomatic improvement. She became afebrile and gained weight to 134 pounds. Films of the chest, however, revealed only slight improvement. Study of the patient was continued in the outpatient clinic. A skin test was done with histoplasmin 1:1000 and found positive. Sputum specimens were examined for histoplasma capsulatum by the Illinois Department of Public Health and found to be positive on the following dates: Feb. 15, 1949, Feb. 24, 1949,

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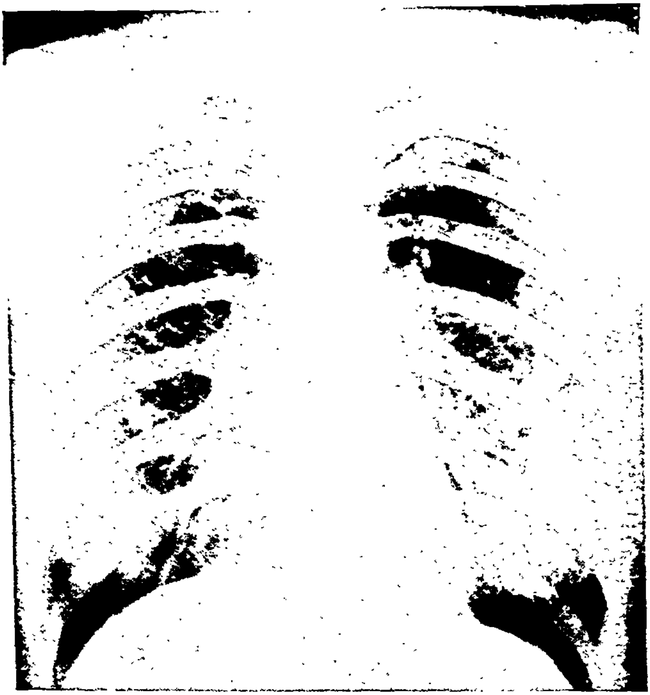

Fig. 1.—Infiltration in right upper lobe, Nov. 13, 1948.

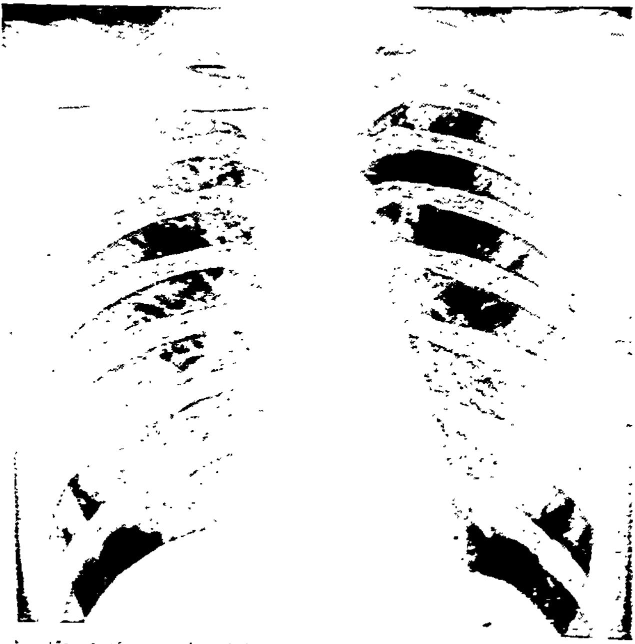

Fig. 2.—Cavitation in the right upper lobe, May 2, 1949, prior to resection.

April 27, 1949, and Sept. 1, 1949. Blood was examined for complement fixing antibodies by Dr. Michael L. Furcolow of the U. S. Public Health Service. This test was positive for histoplasmosis on March 30, 1949, April 19, 1949, May 1, 1949, August 8, 1949, and April 28, 1950. In November, 1950 the test was negative.

During her stay in the sanitarium the patient was treated with a course of penicillin daily for about one month. Streptomycin 1 Gm. daily was given from February, 1949, to April, 1949. In May, 1949, the patient was given aureomycin therapy 2 Gm. daily for about three weeks. Treatment had slight, if any, effect on the roentgenogram appearance of the lesion. Because of this, pulmonary resection was recommended. In February, 1950, a wedge resection of the lesion was done* at the Mayo Clinic with uneventful recovery. Follow-up of the patient to the present time (March, 1951) reveals no evidence of recurrence.

Description of specimen: The lesion measured 6 by 4.5 by 3 cm., and showed cavitation. Histoplasma capsulatum was cultured from the removed specimen. Histologically it was diagnosed chronic granuloma with cavitation.

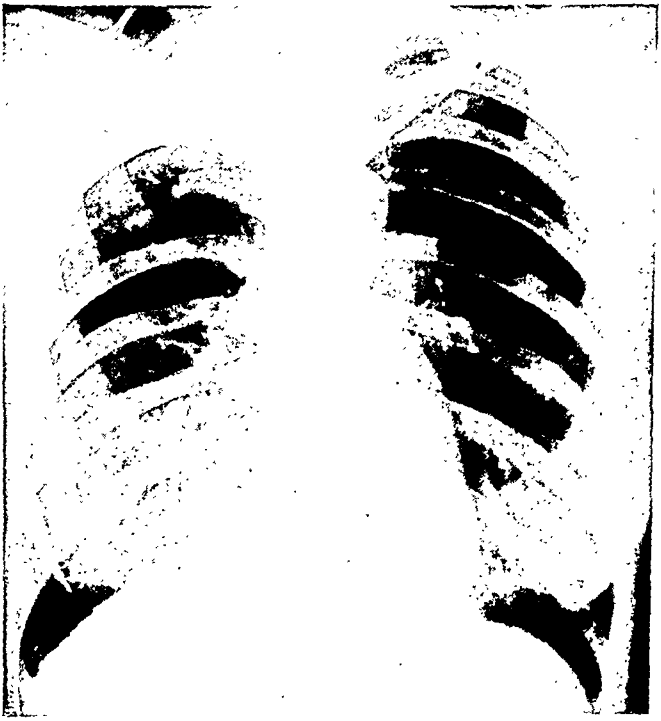

Fig. 3.—Roentgenogram Oct. 27, 1950, following resection.

DISCUSSION

It has become obvious that infection with histoplasma capsulatum may have several different manifestations. The commonest of these is the presence of a positive skin reaction to histoplasmin. Varying percentages are found east of the Rockies, reaching a peak of 80 per cent of persons living in the vicinity of Kansas City. A large proportion of these persons show multiple pulmonary calcifications, frequently larger in size than those associated with primary tuberculous lesions. A careful search for the stage of the disease which precedes these calcifications has revealed that it takes the form of small pulmonary infiltrations, frequently subclavicular, and usually without clinical symptoms. These infiltrates have usually been found in children, although it is probable that they occur in adults also, but are frequently overlooked. Another form of the disease consists of that in which there is dissemination to numerous organs. In this form, the skin test is frequently negative and the outcome usually fatal. The diagnosis is usually made at autopsy. A fourth type of this disease is the destructive but localized type of pulmonary lesion seen in this patient. This has rarely been described, and is easily mistaken for pulmonary tuberculosis.

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*By Dr. O. Theron Clagett.

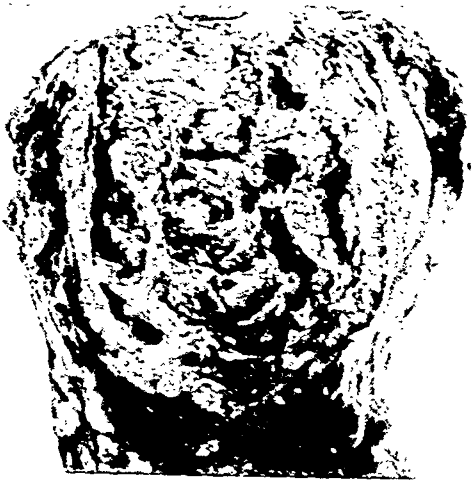

Fig. 4.—Photograph of gross specimen, resected February, 1950.

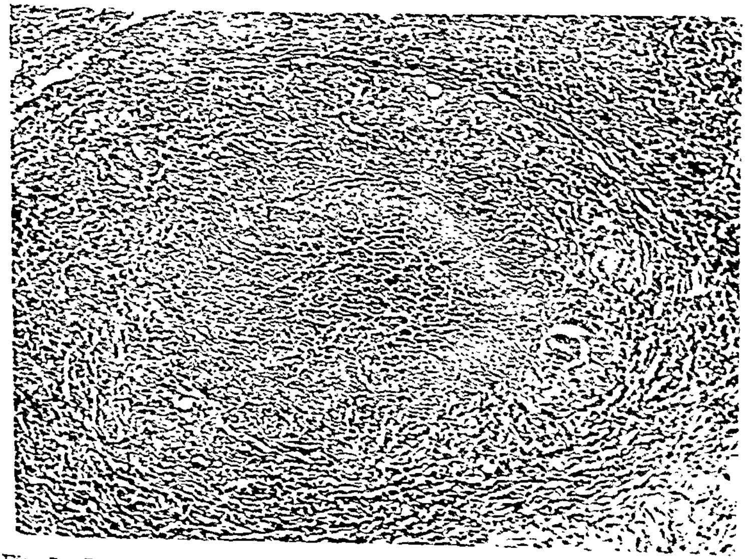

Fig. 5.—Photomicrograph of the lesion, showing granuloma with giant cells.

It appears then that the life cycle of histoplasma capsulatum infection resembles that of pulmonary tuberculosis very closely. It begins with pulmonary infiltrations which are asymptomatic, frequently healing with calcifications, thus resembling primary tuberculosis. Reinfections later in life may take the form of isolated organ histoplasmosis such as the pulmonary lesion described in this paper or the disseminated and highly fatal form, thus again resembling some of the adult type of tuberculosis.

The diagnosis in this patient was aided by the fact that the patient had a negative tuberculin test. It was proved by the presence of a positive histoplasmin test, the repeated finding of histoplasma capsulatum in the sputum and the presence of the complement fixation reaction in the blood serum. It was verified by culture of the organisms from the surgical specimen.

Drug therapy was unavailing, and up to the present time no drug has been described which has any marked effect upon this organism. This renders the treatment by means of resection the only therapy at our command at present.

This case accents the importance of making a careful diagnosis in patients who have lesions resembling pulmonary tuberculosis. Any patient who has a negative tuberculin test must be presumed to be nontuberculous. Failure to find tubercle bacilli after adequate search should be a warning to look thoroughly for other disease.

CONCLUSION

A case of a rarely described type of histoplasmosis, diagnosed prior to surgery, is presented. This was a destructive isolated pulmonary lesion apparently cured by pulmonary resection. The diagnosis was proved by the histoplasmin skin test, the presence of histoplasma capsulatum in the sputum repeatedly, and the finding of positive complement fixation reactions in the serum. The organism was also found in cultures from the surgical specimen. This is believed to be one of the earliest cases to be diagnosed prior to planned surgical resection.

I am very grateful to Dr. M. R. Lichtenstein, Chief of the Medical Service of the Municipal Tuberculosis Sanitarium of Chicago, for his help in preparing this manuscript; to Dr. Michael L. Furcolow, Senior Surgeon of the United States Public Health Service, for laboratory studies; to Dr. O. T. Clagett of the Surgical Section of Mayo Clinic; to the Board of Directors of the Livingston County Sanatorium, Pontiac, Illinois, for permission to use this material; and to the patient who has been most cooperative.

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Original Communications

THE PROGNOSIS OF RESIDUAL BRONCHIECTASIS AFTER INCOMPLETE RESECTION

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ONE hundred ninety-four patients with bronchiectasis had pulmonary resections in Temple University Hospital from 1936 through 1949. We have been able to follow adequately 159 of these one to fifteen years. The response of the patient to surgery is pretty well established within six to twelve months after surgery, so we believe this follow-up study conducted by Dr. Long, who specializes in internal medicine and therefore took no part in the operative procedure, is unbiased and accurate, and has prognostic significance.

Since there is general agreement that bronchiectasis does not markedly influence longevity although it does impair health, we operate in an effort to relieve annoying symptoms and to make living more pleasant. The most important, constant, and annoying symptom of bronchiectasis is cough with expectoration. To justify surgery completely we should be able to offer to the patient some measure of assurance that this symptom will be eliminated with little or no risk to life and without producing or exaggerating dyspnea. The low mortality of lobectomy is well established.

This paper concerns itself with our successes, as well as our failures, in respect to cough with expectoration and, to a lesser degree, dyspnea.

TABLE I. NO COUGH—118 PATIENTS

A. All disease removed (except in 14 patients)
B. Presence or absence of dyspnea depends on pulmonary function
    1. Adequate function
        No dyspnea—71 Patients
    2. Markedly impaired function
        Dyspnea—47 Patients

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

From Temple University Hospital.

TABLE II. COUGH STILL PRESENT—41 PATIENTS

A. All have residual disease
Improved—36
No better— 5
B. Dyspnea is not correlated with function

TABLE III. RESIDUAL DISEASE—55 PATIENTS

	PATIENTS	PERCENTAGE
No cough or symptoms	14	25.5
Less cough, but still present	36	65.5
Increasing cough	5	9.0
Total	55	100.0

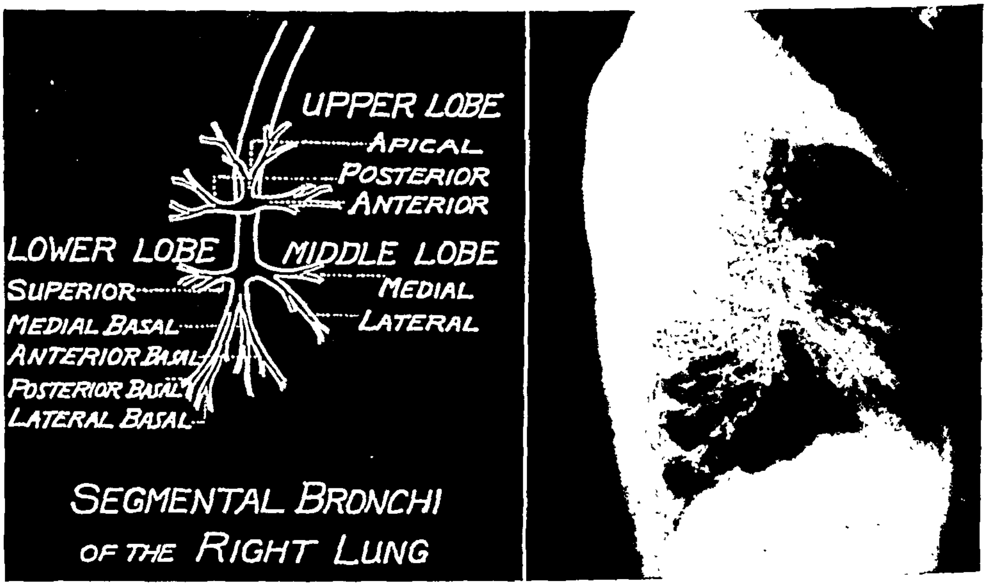

Fig. 1.—Lateral view of bronchogram of normal right lung demonstrating each individual segmental bronchus.

Of the 159 patients adequately followed, 118 are free of cough. Only fourteen of these have any residual disease. In the patients with unilateral bronchiectasis, pulmonary ventilation studies were essentially unchanged by surgery. Many of the patients complained less about dyspnea when infection and sputum were eliminated.

Forty-one patients still have some cough with expectoration, although thirty-six of these are improved. All of these patients have residual disease, and cough

TABLE IV. PATIENTS WITH RESIDUAL DISEASE

	PATIENTS	PERCENTAGE
Bilateral bronchiectasis	48	87
Unilateral bronchiectasis	7	13
Total	55	100

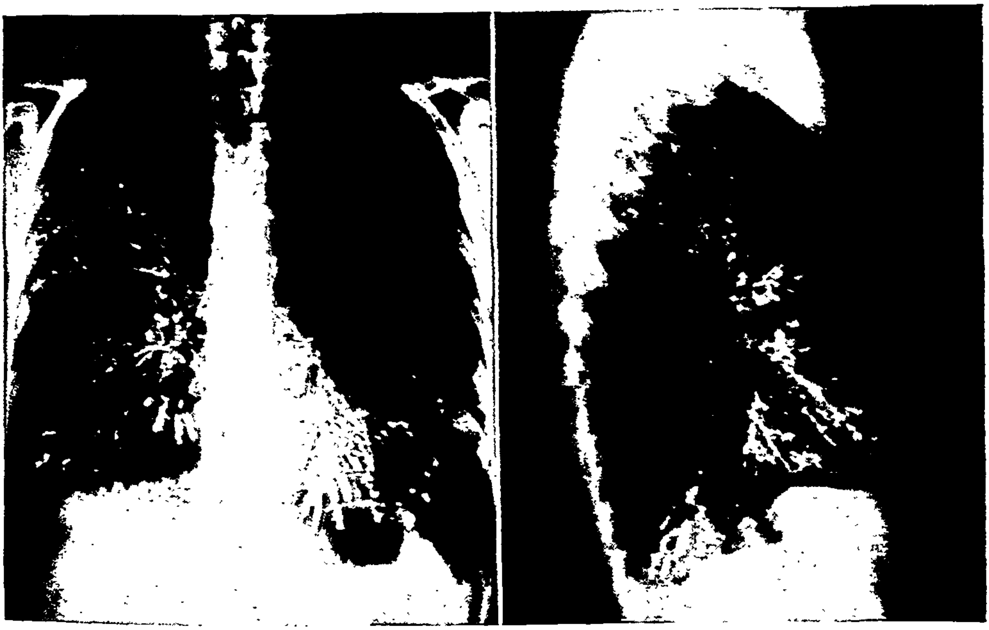

Fig. 2A.—(L.C.) Bronchogram of right lung preceding left lower lobe lobectomy as part of bilateral stage procedure.

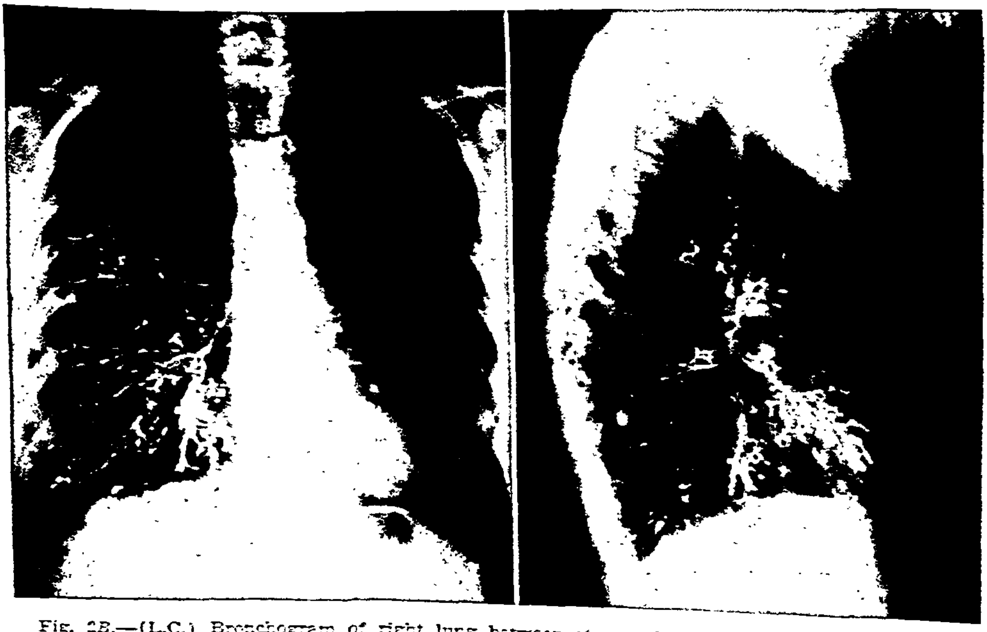

Fig. 2B.—(L.C.) Bronchogram of right lung between stages showing disease in middle lobe missed by original bronchogram.

is, in most instances, proportionate to the degree of residual bronchiectasis. Also, with residual disease, dyspnea is not correlated with ventilation, subjective complaints of dyspnea being out of proportion to the reduction in ventilation.

Of the fifty-five patients with residual disease, 14 (25.5 per cent) have no cough or symptoms of any kind at the time this study was compiled, although, of course, we do not know how long this will last.

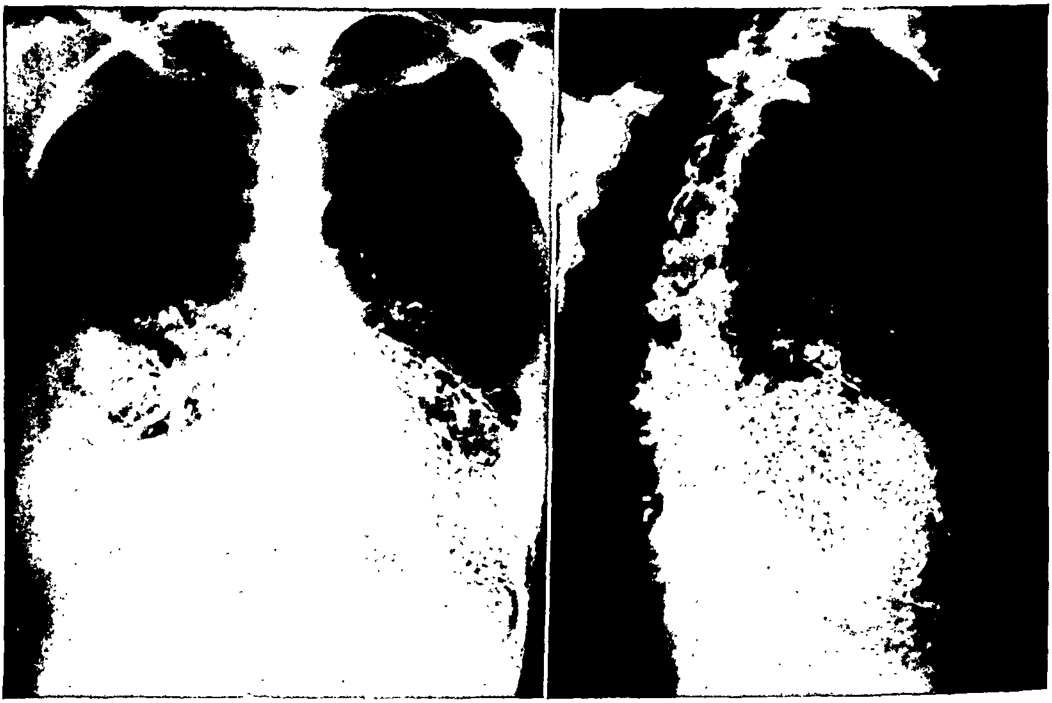

Fig. 3.—(E.W.) This is an example of a too vigorous attempt at saving normal lung. Removal of one more segment on each side would have eliminated cough, and probably would have made little difference in ventilation.

Of the fifty-five patients with residual disease, forty-eight had bilateral bronchiectasis, and only seven had unilateral bronchiectasis. Many of these patients have mild disease producing mild symptoms, but it is also likely that we have tended to be overly conscientious in conserving lung structure when bilateral resection is anticipated.

Thirty-nine (71 per cent) of this group of fifty-five patients had known minimal disease on the opposite side and further resection is being considered.

TABLE V. CAUSES FOR RESIDUAL DISEASE

	PATIENTS	PERCENTAGE
Known minimal disease on opposite side	39	71.0
Judgment at time of surgery	3	5.5
Faulty evaluation	13	23.5
Total	55	100.0

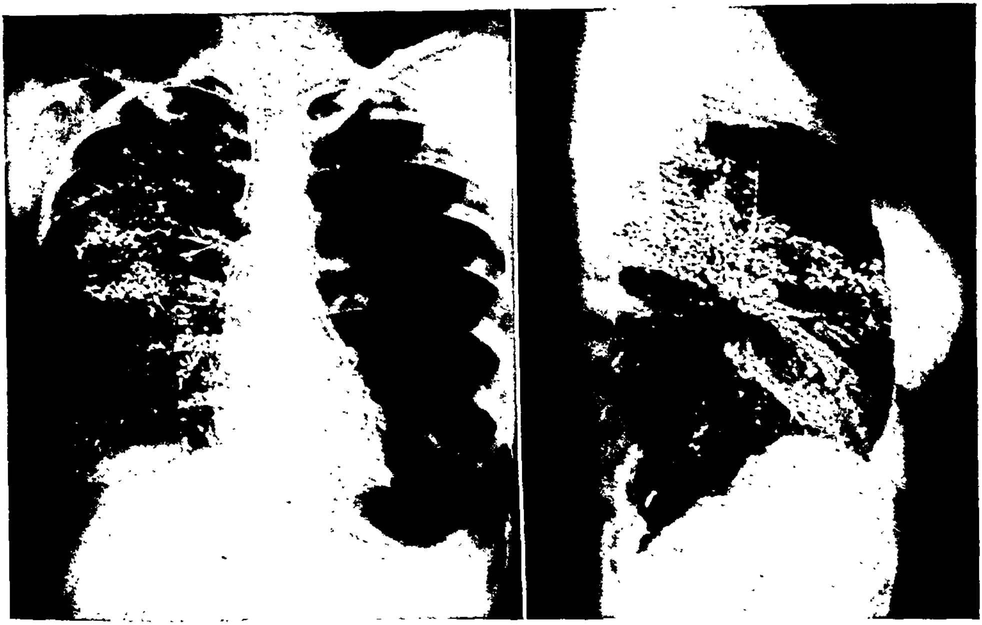

Fig. 4A.—(L.W.) Demonstration of poor filling of superior segment of the right lower lobe on bronchography. Segment was allowed to remain at surgery.

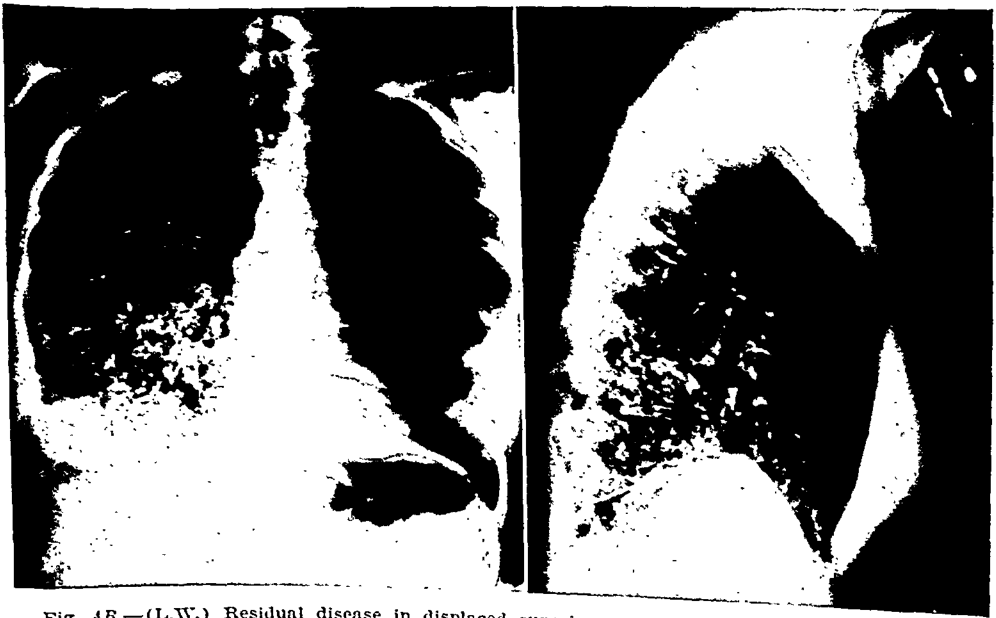

Fig. 4B.—(L.W.) Residual disease in displaced superior segment after basal segments were removed. Symptoms are in proportion to residual disease.

Three (5.5 per cent) have disease *remaining* because it was considered better judgment to leave some less severely diseased lung than to chance removing too much. This judgment we now believe was faulty. It is suspected that patients who are operated on bilaterally do tend to have reduced ventilation, although our series of this type is so small that we hesitate to be dogmatic about this.

Thirteen (23.5 per cent) have residual disease due to faulty evaluation.

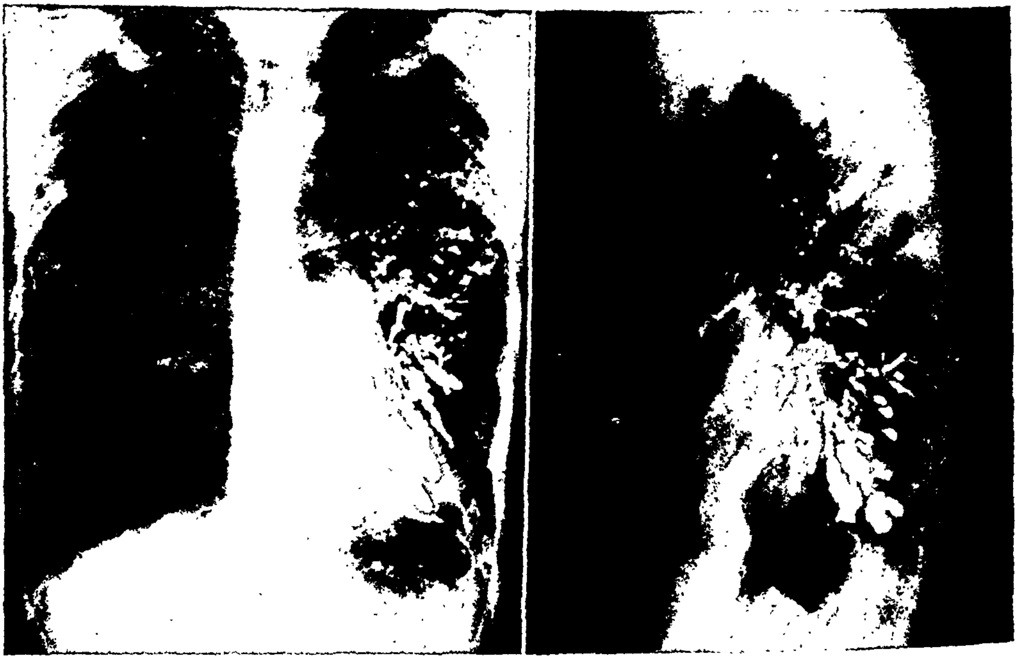

Fig. 5*A*.—(F.H.) Bronchogram of left lung showing saccular bronchiectasis.

These factors may contribute to incomplete resection. Since accurate surgery depends so entirely on bronchography, a faulty bronchogram should not be accepted. Bronchography should be so clear that each segmental bronchus can be identified and alveoli demonstrated (Fig. 1).

Bronchography should be done with the lung as free of secretions as possible. The preparations used will not displace secretions, and iodized oil is so slowly absorbed that after one incomplete mapping with lipiodol, further evaluation is difficult. The water-soluble preparations now available have not proved themselves as yet. The ones we have used have been so viscid that they

TABLE VI. REASONS USUALLY CONTRIBUTING TO RESIDUAL DISEASE

1. Faulty bronchogram
2. Limitations of method of bronchography
3. Attempted evaluation at operation
4. Fear of removing "too much lung"
5. Improper anatomical evaluation of segments
6. Surgical technique

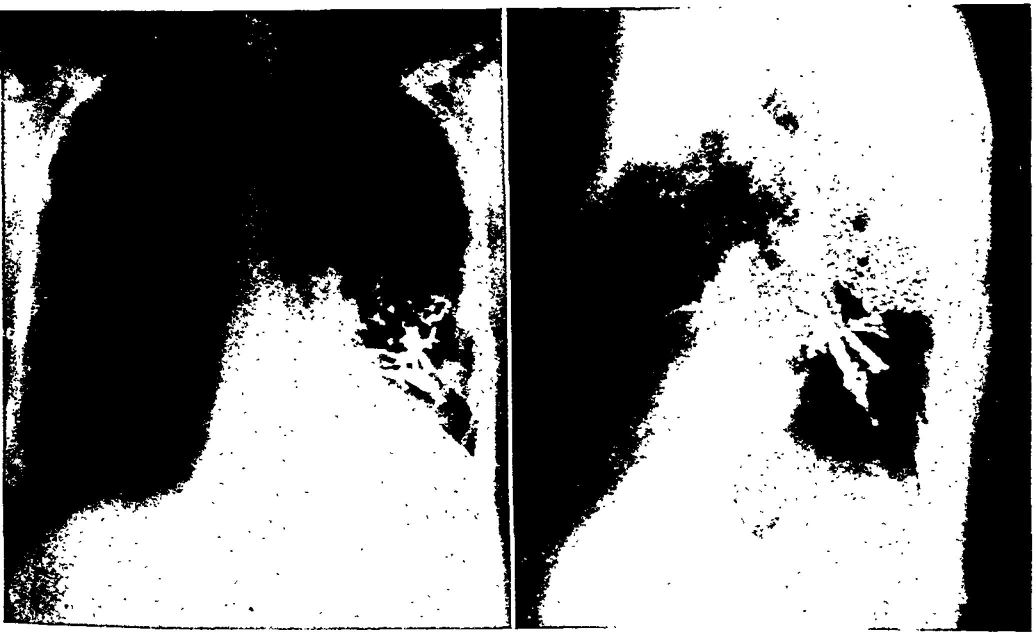

Fig. 5*B*.—(F.H.) Repeat bronchogram of left lung after palliative removal of left lower lobe. As is obvious, considerable disease remains.

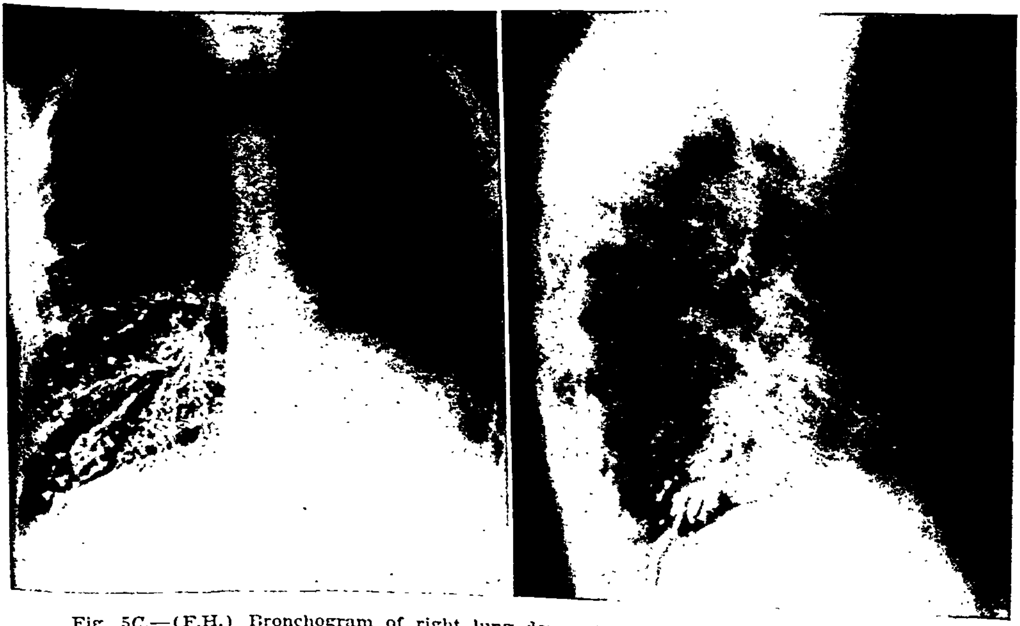

Fig. 5*C*.—(F.H.) Bronchogram of right lung demonstrating disease in right lower lobe.

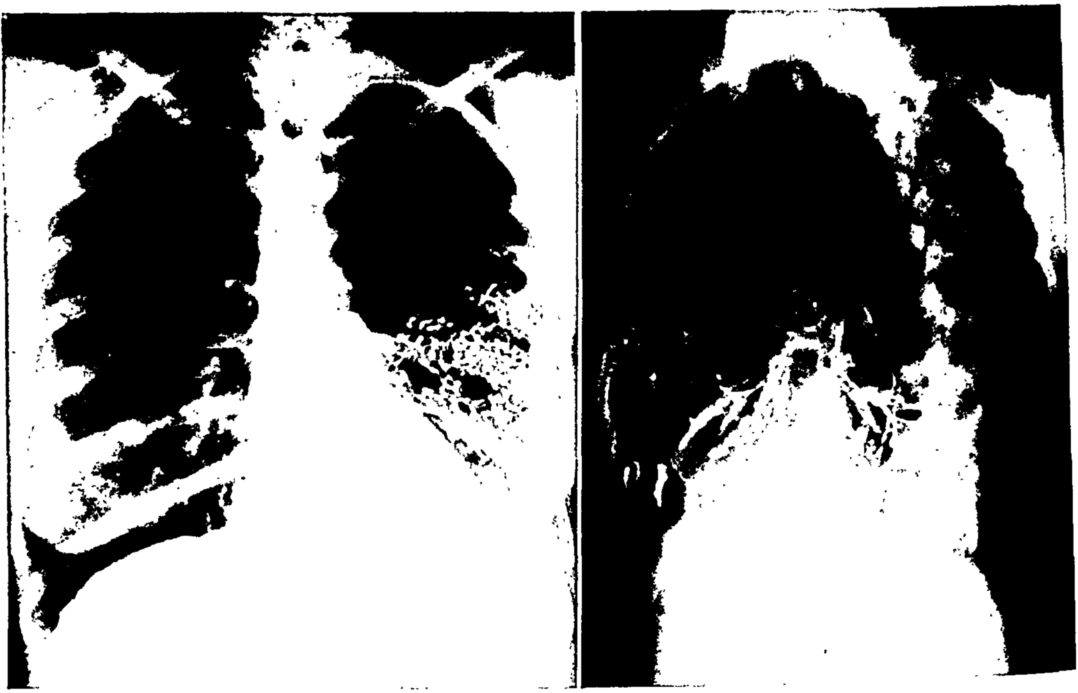

Fig. 6A.—(C.W.) Bronchogram of left lung showing extensive disease.

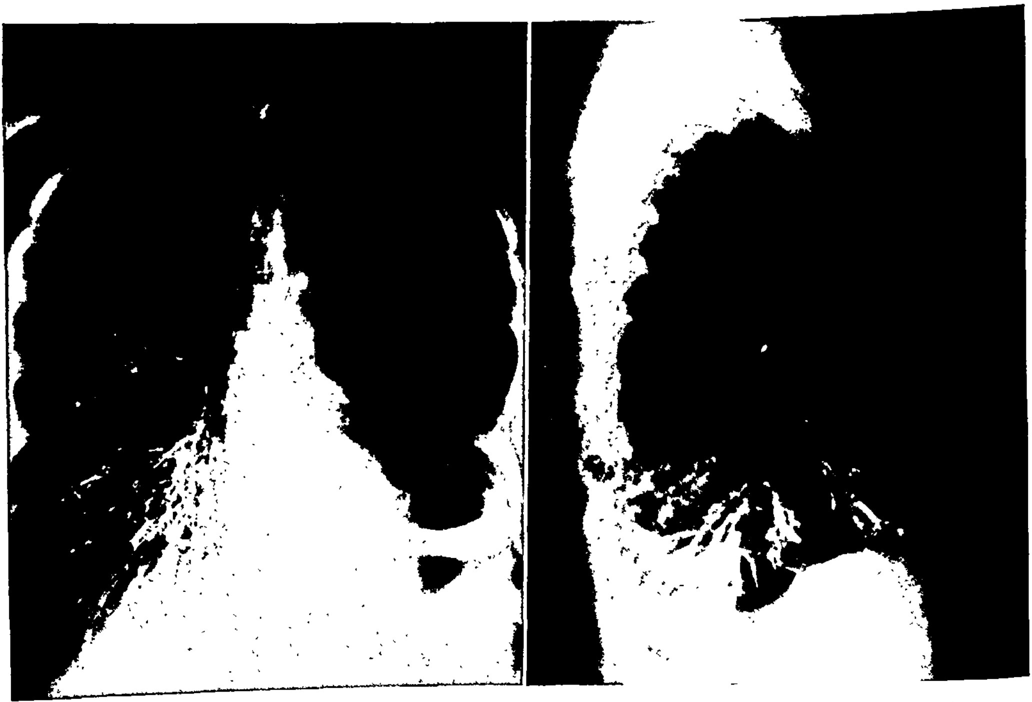

Fig. 6B.—(C.W.) Bronchogram of right lung after surgery on left side. This side shows much less involvement, but the patient was not relieved of symptoms until the disease was eradicated from this less severely affected right side.

do not fill the alveoli well. The rapid absorption of water-soluble preparations, which would allow repeated bronchography as often as necessary to determine the extent of disease, gives us hope that an improved preparation of this nature may be developed soon. We have learned to repeat lipiodol mapping in bilateral cases between stages to avoid being fooled by secretions (Fig. 2 *A*, *B*).

Fig. 3 is an example of shaving too close at operation. One more segment would have eliminated cough and would not have impaired ventilation.

Fig. 4*A* shows poor filling of the superior segment of the right lower lobe. This segment was not removed at operation. Fig. 4*B* shows residual disease in the displaced superior segment. The patient's symptoms are in proportion to the amount of residual disease.

Fig. 5*A*, *B*, and *C* demonstrates a case of saccular bronchiectasis in which palliative resection was done. There is residual disease present because we considered it poor judgment at the time to remove so much lung on one side in the presence of bilateral disease. Evaluation of pulmonary ventilation before and after resection has led us to believe that this reasoning was erroneous. This patient, incidentally, has no symptoms.

The patient whose films are shown in Fig. 6*A* and *B* had very little change in symptoms until the apparently less involved lobe was resected. She is now symptom free.

Since it is impossible to evaluate the extent of bronchiectasis at operation, preoperative bronchography must be accurately carried out. If external spirometry and bronchospirometry are done preoperatively, one has a fairly good means of evaluating the function which can be expected postoperatively. Removal of all bronchiectatic lung is the only way to insure freedom from cough with expectoration.

DISCUSSION

DR. J. H. WALKER, Brookline, Mass.—I agree with the speaker that the total removal of the diseased tissue is necessary in the treatment of bronchiectasis if one is to obtain a good result; but also, to qualify that statement, one should conserve all normal pulmonary segments at the same time in order to achieve the maximum benefit. We have had experience with the surgical treatment of 150 odd cases of bilateral bronchiectasis in the Overholt Thoracic Clinic, and it has been necessary to do bilateral surgery in only one-third of the cases. We have recently brought back a few of the cases for pulmonary function studies to evaluate effects of bilateral segmental resection in severe bronchiectatic cases.

(Slide.) These are four patients that have had extensive bilateral surgery; three of these I am sure could be classified as total respiratory cripples prior to surgery. They had severe cough with sputum averaging from one-half cup to two cups per day. They are now free of cough and purulent sputum. You will note in the first column the total number of segments that were removed varied from seven to eleven. The girl who had eleven segments removed had segments removed from all five lobes. The percentage of predicted maximum ventilation postoperatively ranged from 48 to 81 per cent for total pulmonary function. This is a nice surprise, and is better than we had anticipated with bilateral excisional therapy.

(Slide.) This is a breakdown of the number of segments removed from each lung and also the differential bronchospirometry. There is a fairly close correlation here between the number of diseased segments removed, the number of normal segments left behind, and the proportion of ventilation carried by each lung. This emphasizes not only the necessity of removing diseased segments, but also the preservation of all normal pulmonary segments.

DR. J. GORDON SCANNELL, Boston, Mass.—As a footnote to the paper by Dr. Rosemond, I would like to present the by-product of a recent study we have conducted at Massachusetts General Hospital on what happens to patients a long time after the diagnosis of bronchiectasis has been made. Looking back in our records we find that in 1939-1940, there were some eighty-seven patients admitted in whom a diagnosis of bronchiectasis was made. Of this number forty-three were operated on, but there was a small group of nineteen in whom the diagnosis was made and in whom the anatomic indications for resection were present, but in whom, for one reason or another, operation was not carried out. In the past month I have been able to trace seventeen of these; of the seventeen, eleven are in excellent health. Four—and this was rather surprising to me—died of fatal massive pulmonary hemorrhage; and one died of general decay which was obvious when he was in the hospital. It is interesting that recently none of the patients have died of sepsis, and it is obvious also that penicillin had a tremendous modifying effect in the past few years.

DR. BURNETT (Closing).—In closing this discussion I simply want to remind us all of the fact we have recognized so often; that impressions gained are frequently dispelled by a little analysis. When this analysis was made, I was surprised at the number of patients in our group who had had residual disease. I would not have believed that from the impression gained as we went along. Second, I would like to emphasize the value of palliative resection in extensive disease. We have not tackled any patients with five lobes involved, as Dr. Walker and Dr. Overholt have done; we have not had quite that much courage, but segmental resection certainly makes this possible. We have, however, utilized segmental resection in a high percentage of these patients.

One of the most extreme examples of palliative resection was one young lady from South Africa who had five lobes involved, who had very advanced disease in the left lower lobe, and moderate to advanced in others. After removal of the left lower lobe and lingula her symptoms were decreased by about 90 per cent, which she felt was quite worth while, and we agreed. Moderate involvement on the opposite side to that which is symptogenic and saccular certainly warrants conservatism on the opposite side, as Dr. Rosemond has demonstrated. We believe it may be well to observe those patients and see if symptoms warrant further attack. As you saw, some fourteen of those patients, although having residual disease, had no symptoms whatever, and the others were markedly improved, with the exception of five.

I want to thank Dr. Walker and Dr. Scannell for their discussion, and to say that we will gather great impetus from their suggestions.

RADICAL PNEUMONECTOMY

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NEW YORK, N. Y.

THE purpose of this report is to submit data showing that a lung can be removed in a block dissection together with most of its regional hilar and mediastinal lymph nodes.

The technical steps and the clinical implication of the procedure to be discussed are based upon an experience of thirty-nine such operations successfully performed on the Thoracic Service of Memorial Hospital.

DEFINITION

The technique of lung removal for primary carcinoma of the lung has been described many times since the first successful one-stage pneumonectomy in 1933.[6] However, although rapid progress has been made in the management of the complete physiologic and anatomic problems inherent in this procedure, there remain several confusing and controversial features which require clarification.

In the literature the term, "pneumonectomy," has been employed either alone or with the adjectives, "simple," "total," or "dissection." These terms have been used interchangeably and, as a consequence, there is some confusion as to whether an actual difference exists. When some of the hilar or mediastinal lymph nodes have been included in the procedure, these have been alluded to for the most part in indefinite terms as to their location or whether they were individually excised or taken en bloc with the lung.[12] For over two years we have been distinguishing the removal of a lung alone—or simple pneumonectomy—from cases in which mediastinal node dissection is included. During this time we have been impressed, in concurrence with the views expressed by Allison[1] last year, with the need for an inclusive term for the more radical procedure. It is suggested, therefore, that these operations for lung cancer be defined as follows:

A *simple* pneumonectomy is the excision of the lung alone.

A *radical* pneumonectomy is the excision of the lung in continuity with its regional lymph nodes located in the hilar and mediastinal areas.

When adjacent structures are included in the procedure they may be added as follows: e.g., a "simple pneumonectomy with resection of ribs 3, 4, and 5"; or a "radical pneumonectomy with a partial pericardiectomy."

The desirability of removing a malignant tumor en bloc with its regional lymphatic drainage area is an established principle of cancer surgery when certain circumstances exist:

From the Thoracic Surgical Service, Memorial Hospital.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

1. No distant metastases are discernible;
2. No other form of therapy has any real value;
3. The morbidity and mortality rate of the surgical procedure required remains reasonably low;
4. From experience, a high index of probability exists for present or future involvement by cancer of the lymphatic pathways to be excised;
5. If these lymphatic areas are not resected at the time of the original procedure, they would be hidden within the body so that their future involvement by metastases would escape early detection.

These are principles that have been applied to the treatment of cancers arising in the colon, stomach, and breast.

It is known that the hilar and mediastinal lymph nodes can become involved by metastases from a primary carcinoma of the lung. Ochsner[8] reported in 3,047 collected cases of carcinoma of the lung that these nodes were involved in 72.2 per cent. His relative distribution of lymph node metastases in 1,298 collected cases showed that 69.7 per cent were in the tracheobronchial group, 20.7 per cent in the abdominal, 17.4 per cent in the cervical, and 4.2 per cent in the supraclavicular areas.

Figures from Koletsky's[7] series of 100 autopsies support the fact that regional lymph nodes are indeed the chief site of early metastasis for most lung carcinomas.

In consideration, then, of the traditional treatment of cancer for other sites and the assembly of pathologic facts as listed above, it would seem that any practice of plucking out a lymph node here and there "like raisins from a bun" in these areas should be discouraged if a method can be devised to excise them in continuity with the primary tumor. It is felt that by a relatively detailed anatomic and physiologic description of pulmonary lymphatic drainage, the possibility and techniques of such a block dissection will become apparent and appear reasonable.

LYMPHATIC SYSTEM OF THE LUNG

No article on lymph node drainage should begin without a tribute to the excellent studies of Rouviere[10] and it is strongly recommended that his original work be consulted for a more detailed and thorough account than that which follows and which was derived liberally from it. Rouviere points out that even as there are different anatomic features to the contents of the right and left thoracic cavities, so are there variations in the lymph node pattern and drainage on both sides. He defines several major divisions of node-bearing areas; mediastinal, peritracheobronchial, and intrapulmonary.

MEDIASTINAL LYMPH NODES

The anterior mediastinal, or prevascular nodes, are placed in the superior portion of the mediastinum in front of the large blood vessels, which are connected with the heart. On the right side, the chain is placed in front of the superior vena cava and the right innominate vein in a pre-venous location with one prominent node at the junction of both innominate veins. Lymph nodes about the right phrenic nerve and inferior vena cava also drain into this pathway, terminating at the right jugulo-subclavian junction or the right lymphatic duct.

The left anterior mediastinal chain is pre-arterial, or, more exactly, pre-aortico-carotid. It commences in a node of good size placed just in front of the ligamentum arteriosum. which rests on the left pulmonary artery. The pre-aortico-carotid chain (left anterior mediastinal chain) ascends between the phrenic and the vagus nerves. following chiefly the course of the latter nerve. The nodes of this chain are placed from below upward. at first in front of and on the superior border of the aortic arch. then above this vessel on the anterolateral surface of the common carotid artery. Quite often a few nodes are placed somewhat medially along or a little in front of the right lateral border of the thymus gland. The left lymphatic mediastinal path terminates in the thoracic duct or directly into the jugulo-subclavian venous junction.

The transverse anterior mediastinal chain nodes are placed along the superior and inferior borders of the left innominate vein and may form an anastomosis between the right and left mediastinal pathways. as well as between the right and left paratracheal chain of nodes.

The *posterior* mediastinal nodes are juxta-esophageal. Normally 2 to 5 nodes are placed along the lateral borders of the esophagus at the level or somewhat below the inferior pulmonary vein and occasionally between the esophagus and the aorta. The greater number of the efferent vessels from these pour their contents into the subcarinal (inter-tracheobronchial) nodes while the rest empty into the thoracic duct.

PERITRACHEOBRONCHIAL NODES

These include the paratracheal nodes. subcarinal (or nodes of the bifurcation). and the nodes of the pulmonary roots. Two principle chains of nodes are present: the right and the left lateral or paratracheal chains and a few inconstant retrotracheal nodes.

a. *Right.*—The paratracheal nodes are placed in a niche. the boundary of which is as follows: in the front. the superior vena cava and the right innominate vein behind. the right anterolateral surface of the trachea; medially. the arch of the aorta and the innominate artery: above. the right subclavian arteries surrounded by the right recurrent nerve: below. the arch of the vena azygos; laterally. the niche is closed by that part of the mediastinal pleura which extends from the superior vena cava and the right innominate vein to the right lateral border of the trachea. and which is skirted by the vagus nerve.

The right paratracheal chain consists of three to six nodes which may be arranged in a single file. although most of these are in groups of two to three in juxtaposition. The chain is not truly vertical but bends somewhat posteriorly. The highest node is at the same time the most laterally placed and the most posterior: and is quite often remarkable for its size. particularly when it receives a large lymphatic trunk coming from the nodes at the bifurcation. It rests immediately below the right subclavian artery but may insinuate itself partly behind this vessel. Above this node arise the nodes of the right recurrent chain. Generally, the lowest node of this right paratracheal chain is the largest and is always found at the arch of the vena azygos, which in the majority of cases covers its lower pole, or actually the node may project over this vein. It has been called the "node of the arch of the vena azygos."

Afferent and efferent lymphatics in this group form a very important ascending right paratracheal lymphatic path, which is a continuation of the vessels which issue from the right lung, the right supra- and retrobronchial nodes, and from the nodes of the bifurcation. This path also receives directly some lymphatic vessels from the trachea, the esophagus, and the thymus gland. It terminates above either in the right lymphatic duct or in a separate entrance into the jugulo-subclavian angle.

b. *Left.*—The *left* lateral paratracheal chain conists of 4 to 5 nodes placed *along the anterior and medial aspects of the vertical segment of the* left recurrent nerve, occupying a position along the recurrent nerve lateral to the left posterolateral border of the trachea, medial to the pleura and medial and posterior to the arch of the aorta and the left subclavian artery. They ascend in the left paratracheal lymphatic path.

The retrotracheal nodes are placed behind the inferior part of the trachea on the path of the lymphatic vessels which unite the subcarinal nodes to the right paratracheal chain. These are present in about one-eighth of the cases. Generally there is but one node placed a little above the tracheobronchial angle near the right border of the posterior surface of the trachea along the esophagus and the vagus nerve.

SUBCARINAL NODES (NODES OF THE TRACHEAL BIFURCATION)

This group occupies the interval between the trachea formed by the divisions of its two bronchi and above the inferior pulmonary veins. There are usually 3 to 5 nodes which are fused in such a way as to have a mammiliform shape indicating an incomplete division of this node into smaller elements. The total number of elements in it varies. These nodes are in contact with the inferior border of the trachea and the suprajacent bronchus. They even extend somewhat in front of the tracheal spur, and there is a bond of union between the subcarinal nodes and the inferior extremities of the paratracheal chain. They are in relation with the pericardium in front and with the esophagus, vena azygos, and aorta behind. The efferent lymphatics from these nodes usually proceed to the right paratracheal chain (rarely to the left paratracheal chain), and reach their destination and pass either in front of or behind the tracheobronchial tree.

NODES OF THE PULMONARY ROOTS

A group of nodes is placed between the elements which make up the pulmonary root. They are listed as anterior, posterior, superior, and inferior, according to their position and relation to the respective bronchus. They are present if anterior in pre-venous, pre-arterial, and pre-bronchial locations. The posterior nodes are found more frequently on the right side than on the left. The superior nodes, about 1 to 3 in number, are found most often on the left side, lie along the superior border of the bronchus, and behind the left pulmonary artery. This chain is constant and continuous above with the lateral tracheal, left recurrent chain of nodes by means of a lymphatic vessel which passes below the arch of the aorta and the ligamentum arteriosum (Fig. 1). There is a node of the loop of the left recurrent nerve at this level. Arnstein attributes certain cases of paralysis of the left recurrent nerve to tuberculosis of these lymph nodes. This might also be true when metastases are present, which burst the nodal capsule and invade the nerve secondarily.

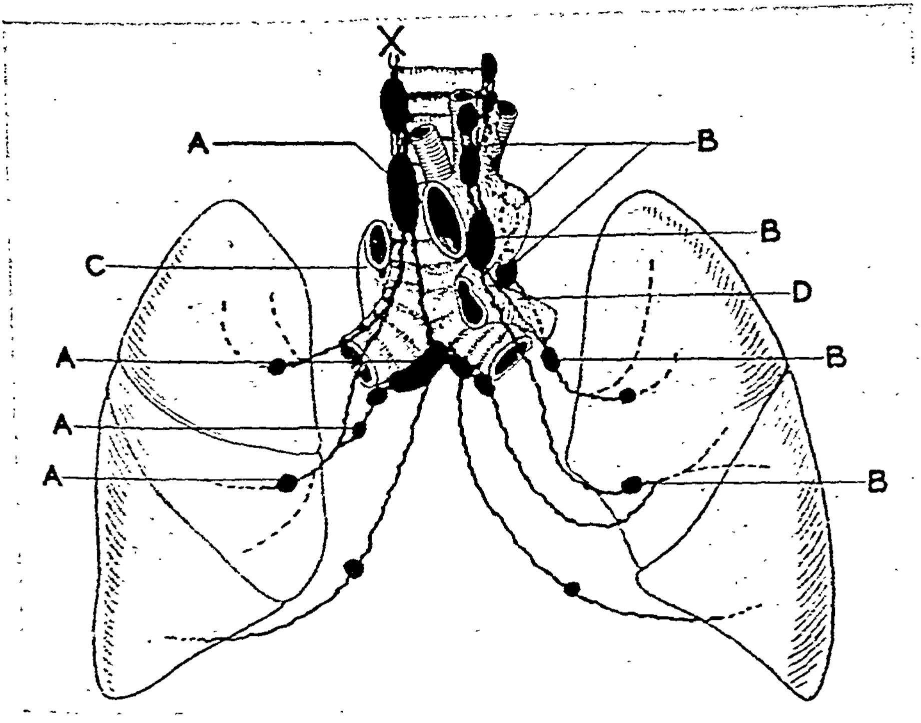

Fig. 1.—Diagram illustrating lymphatic drainage areas of the lungs. These are divided into superior, middle, and inferior regions. The right lung drains entirely up the right lymphatic pathway. The lower two segments of the left lung drain predominantly to the subcarinal nodes and then up the right lymphatic pathway. Occasionally, they may also travel up the left lymphatic pathway, joining the drainage from the superior section of the left lung. (Note that the sections are not confined by major fissures.) Drinker after Rouviere.

The *pre-esophageal* nodes of the inferior pulmonary ligament extend from just below the bronchus to the diaphragm, and are found more often on the left than on the right side (the latter in only 20 per cent of the cases according to Rouviere). These drain to the subcarinal nodes.

INTRAPULMONARY NODES

Nodes are placed along the broncho-arterial trees of each lobe, and it is important to note that the principal node draining one lobe group may rest against the bronchus of an adjacent lobe.

Each lung has three principal lymphatic regions—superior, middle, and inferior (Fig. 1). These are not limited by the usual anatomic boundaries or major fissures.

Right Lung.—The superior lymphatic region is represented by the anteromedial portion of its upper lobe. Its collecting vessels are tributaries solely of the right paratracheal nodes, particularly of the node of the arch of the vena azygos. The middle lymphatic region which includes the posterolateral region of the upper lobe, all the middle lobe, and the superior part of the lower lobe conveys lymph to both the right lateral tracheal and subcarinal nodes. The inferior lymphatic region comprises the inferior region of the lower lobe; the lymph from this region is poured entirely into the subcarinal nodes.

Left Lung.—The superior lymphatic region is comprised of the superior region of its upper lobe, the lymph collecting vessels of which connect with the left paratracheal chain, the node of the ductus arteriosus with the left anteromediastinal nodes and with the node of the loop of the left recurrent nerve. The middle lymphatic region includes the inferior region of the upper lobe as well as the superior and middle regions of the lower lobe. The vessels terminate in part superiorly in the anterior mediastinal and left paratracheal nodes, and in part inferiorly in the subcarinal nodes. The inferior lymphatic region represented by the inferior part of the lower lobe drains to the subcarinal nodes.

Since the subcarinal nodes normally convey their efferent lymphatic trunks to the right paratracheal group, it follows that only the superior region of the left lung in its entirety, and the middle region of the same lung in part, divert their lymph into the anterior mediastinal and left paratracheal nodes, and thence into the left jugulo-subclavian confluence. The remainder of the left lung, part of the middle region, and the entire right lung are finally tributaries of its right paratracheal chain and the right jugulo-subclavian venous confluence. It becomes possible to foretell to some degree by which lymphoid echelon a lesion might progress, the exact seat of which in the lung has previously been established.

LYMPHATIC DRAINAGE OF THE LUNG

Confirmation of Rouviere's dissection and deductions is found in Drinker's[5] report (Fig. 1). He cannulized separately the right lymphatic and the thoracic ducts in dogs. Carbon particles were then deposited carefully by bronchoscope in various segments of the lung and their reappearance in the lymph flow of the right or left cannulas noted.

As might be anticipated from Rouviere's work, when the particles were placed in any segment of the right lung or in the lower two-thirds of the left lung, the lymph emerging from the right cannula (*x* in Fig. 1) was colored by them, while that from the thoracic duct remained clear. Only when particles were deposited in the superior portion of the left upper lobe did they appear in the lymph issuing from the left cannula.

These experiments have found clinical confirmation. It has been noted that metastatic lymph nodes located in the right supraclavicular region just behind the clavicular head of the sternocleidomastoid muscle can be traced to lung carcinomas having their origin in the right lung or in the middle and lower lymphatic regions of the left lung. In addition, the surgeon has a unique opportunity to observe at the time of exploratory thoracotomy, the intervening channels between a primary carcinoma and the highest apparently involved lymph node. This may be graphically demonstrated in the future even in unresectable cases when these areas are delineated by the use of dyes according to the technique of Weinberg and Greaney.[13]

Specimens removed by radical pneumonectomy with their lymph node groups intact and clearly marked so as to maintain their relative position will aid further in delineating the pattern of metastatic spread.

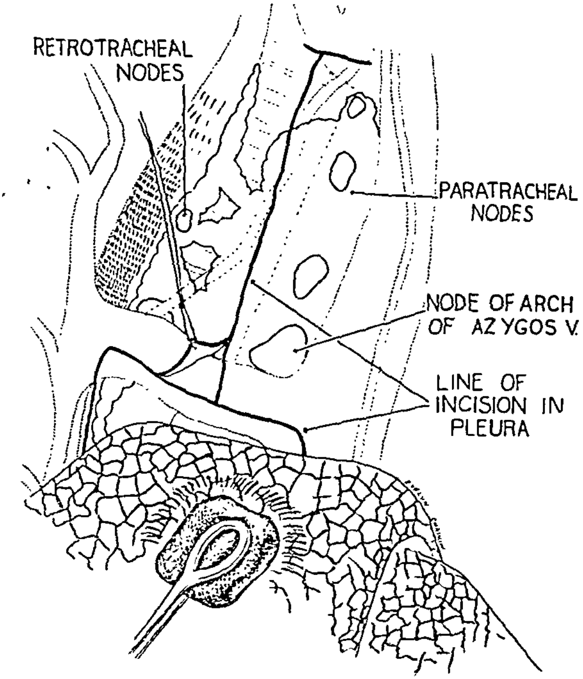

Fig. 2.—Right side view of paratracheal lymph nodes and superior mediastinal pleural incision.

Finally, a careful study of metastatic lymph node involvement from autopsy specimens of patients who have died of lung cancer has already provided, and will continue to provide a real aid in determining the flight of metastases. In this connection, Crafoord[4] reported in his early articles at least three autopsies on patients who had had what was probably a simple pneumonectomy that revealed residual metastatic lymph nodes located in the paratracheal regions.

To be sure, variations in the pathways described above occur. In particular, metastasis from the lower two-thirds of the left lung may travel directly up the left side instead of by the more usual *contralateral spread.*

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These experiments have found clinical confirmation. It has been noted that metastatic lymph nodes located in the right supraclavicular region just

Inferiorly, a horizontal incision follows the azygos vein to the phrenic nerve and thence inferiorly along this nerve to the inferior pulmonary ligament. Posteriorly, the incision of pleura is just anterior to the azygos vein all the way to the diaphragm where it meets the anterior incision.

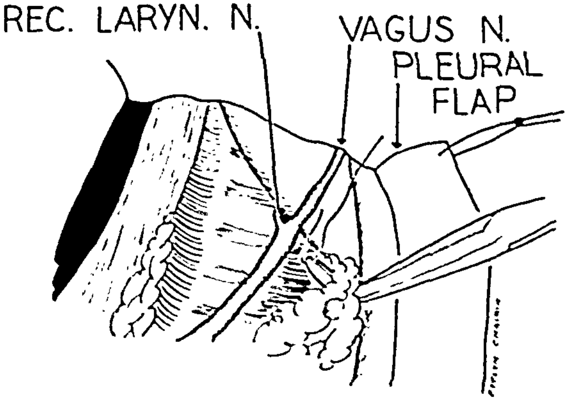

Fig. 4A.—Right side: Apex of dissection beneath right subclavian artery.

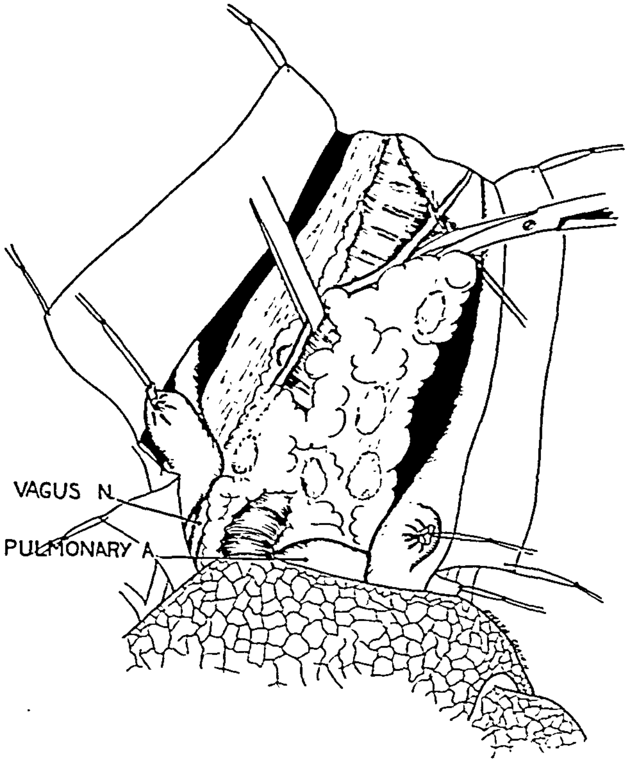

Fig. 4B.—Right side: Retraction of vagus nerve and dissection of paratracheal node-bearing tissue from it. Note clearing of retrocaval space.

In addition, with the choking of one node by metastasis, retrograde extension along pathways not ordinarily taken may occur. Skipping of lymph nodes by metastases has also been noted. By and large, however, metastases follow the echelons outlined above.

This anatomic and pathologic knowledge suggests that removal of the described mediastinal lymph pathways with the lung will improve end results. One may speculate, however, that primary lesions of the lower two-thirds of the left lung will have a poorer prognosis because contralateral lymph node metastases will preclude block dissection.

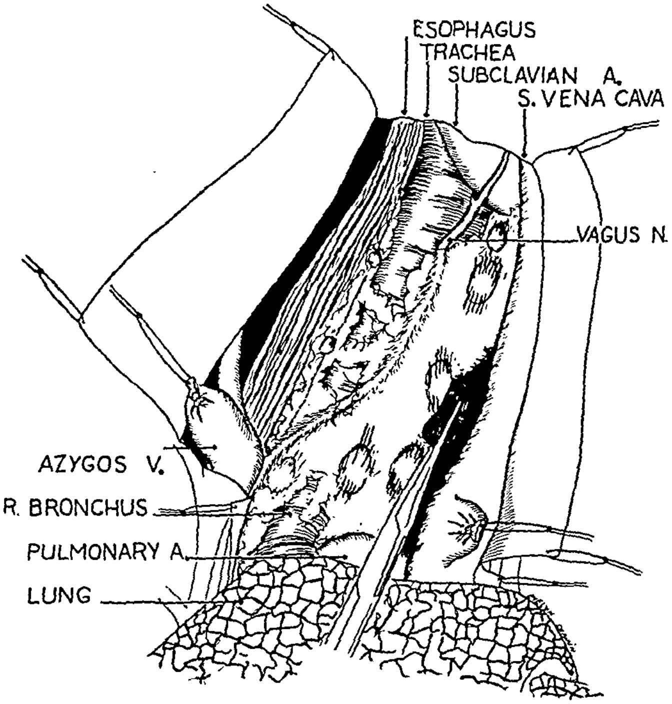

Fig. 3.—Right side: Pleural flaps elevated. Azygos vein divided. Dissection begun beneath superior vena cava.

TECHNIQUE

To approach either lung the patient is placed in a true lateral position and the chest entered through a long parascapular incision and the bed of the fifth rib. The pathologic setting is explored and, if a radical pneumonectomy seems feasible, and the patient's general condition is satisfactory, the procedure is carried out usually in the following sequence.

Right Side.—

1. *Incision* (Fig. 2): The superior mediastinal pleura is incised from the apex of the chest to the azygos vein arch just anterior to the vagus nerve. Superiorly, a short horizontal limb is used to facilitate exposure of the subclavian artery and right recurrent laryngeal nerve.

Inferiorly, a horizontal incision follows the azygos vein to the phrenic nerve and thence inferiorly along this nerve to the inferior pulmonary ligament. Posteriorly, the incision of pleura is just anterior to the azygos vein all the way to the diaphragm where it meets the anterior incision.

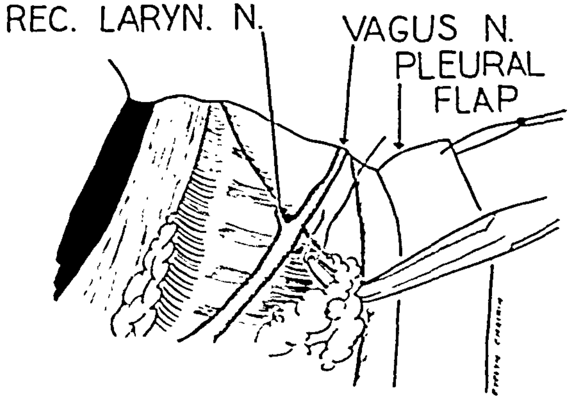

Fig. 4A.—Right side: Apex of dissection beneath right subclavian artery.

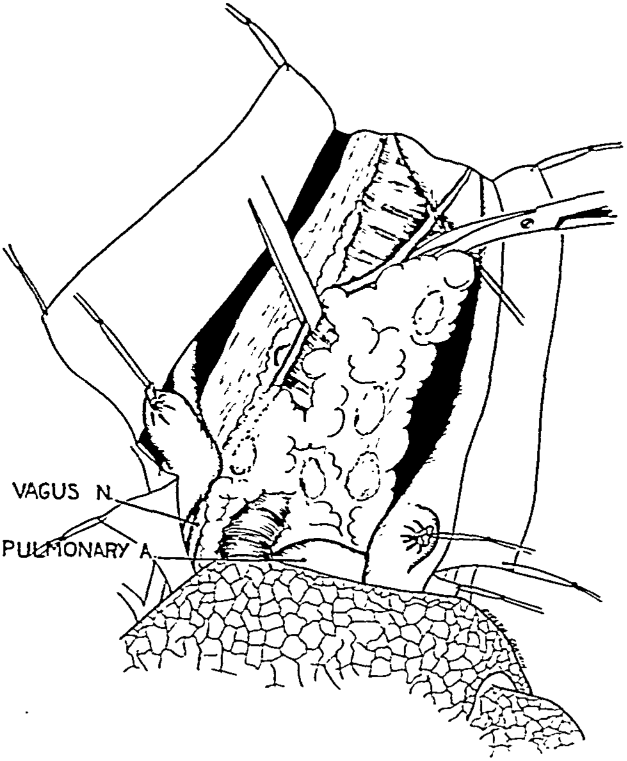

Fig. 4B.—Right side: Retraction of vagus nerve and dissection of paratracheal node-bearing tissue from it. Note clearing of retrocaval space.

In keeping with the tradition of starting a block dissection for cancer with the distantmost points of potential metastasis, the first section of the procedure progresses *from the most peripheral nodes to the hilum.*

2. *Paratracheal dissection* (Fig. 3): The anterior pleural flap is elevated to the anterior surface of the superior vena cava, phrenic nerve being displaced with it. The posterior flap is elevated to the vertebral bodies, leaving vagus nerve uncovered. These flaps are retracted by the weight of Halsted clamps fastened to sutures attached to their free edges.

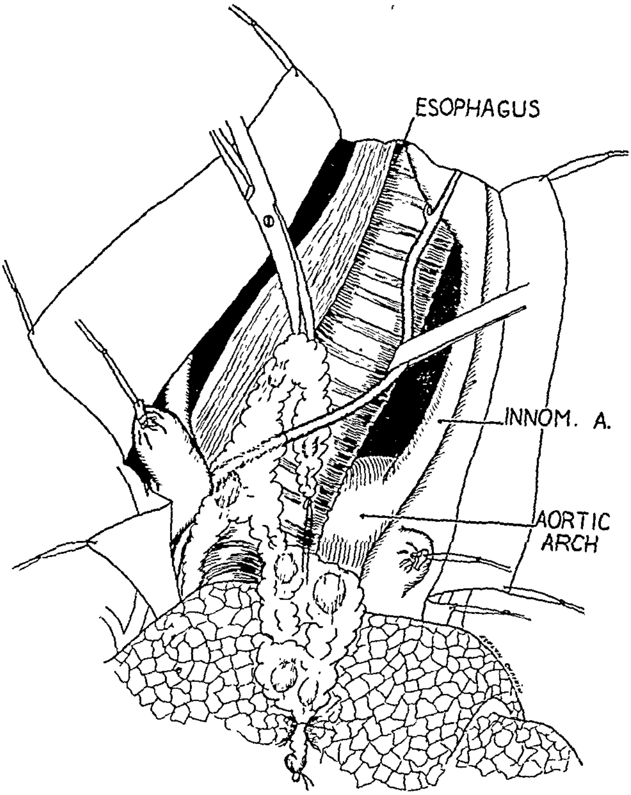

Fig. 5.—Right side: Retrotracheal lymph node dissection and vagus nerve elevated to pass this group beneath it. Note the most constant node at the tracheobronchial angle. Also the boundaries of the retrocaval space.

The azygos vein is transected between ties and ligatures, and the anterior limb lifted forward, thus also elevating the superior vena cava. The areolar tissue is cleared laterally off the latter vein, and its posterior surface freed.

The vagus nerve is mobilized, and superiorly its recurrent laryngeal branch is usually identified. (Fig. 4 [insert]).

The apex of the dissection is the *inferior border of the subclavian artery* where a tongue of areolar, node-bearing tissue is isolated and divided. Next (Fig. 4), attention is turned to the trachea and the fine connective tissue on its right lateral and anterior surfaces which is found to separate readily allowing the whole packet of tissue *anterior to the trachea to be swept downward off* innominate artery and aortic arch to the level of the pulmonary artery. This can usually be done with division of only one or two small vessels. This packet of tissue represents the right paratracheal lymph node chain. To maintain the integrity of this block of lymphatics, the dissection must be carried immediately adjacent to the large vessels and the trachea.

The areolar tissue posterior to the trachea and on the esophagus (Fig. 5) is then freed from these structures, commencing superiorly, dissected downward, and passed under the vagus nerve which is elevated by a Penrose drain. If desired, this last structure can be sacrificed below the recurrent nerve origin.

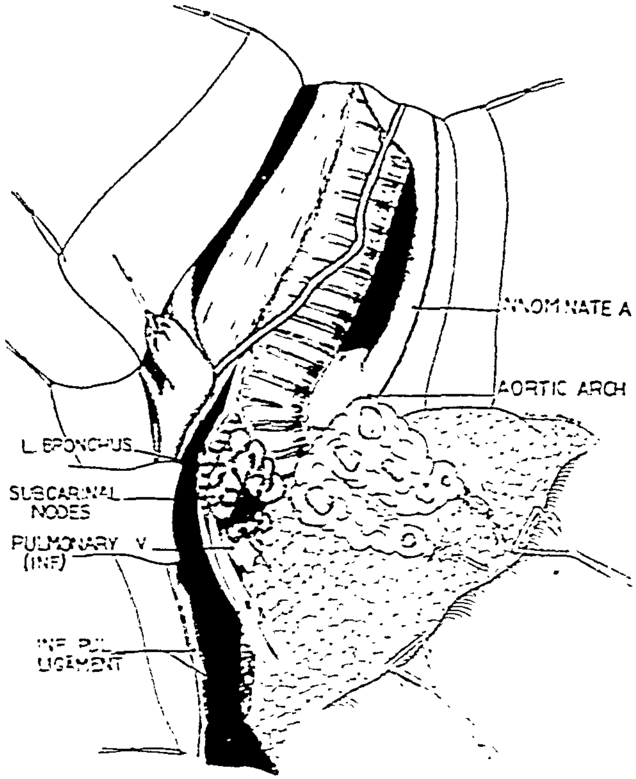

Fig. 6.—Right side: Pre-esophageal nodes have been dissected with the inferior pulmonary ligament. Traction on the lung and on the esophagus exposes the subcarinal lymph nodes. Dissection begun at contralateral bronchus.

Included in this dissection is the particularly important, constant, and large node of the azygos arch lying just above the pulmonary artery. It is mobilized as the dense connective tissue superior to the pulmonary artery and behind the superior vena cava is incised. This paratracheal node packet remains attached to the lung by peribronchial lymphatics and areolar tissue. As each packet of nodes—paratracheal, pre-esophageal, and subcarinal—is mobilized it should be tacked to a convenient point on the visceral pleura to keep them from obscuring the later dissection. So far the nodes placed in front of the right innominate vein and superior vena cava have not been included in our dissection, but probably should be.

3. *Inferior pulmonary ligament dissection* (Fig. 6): The vessel at the lower end of the inferior pulmonary ligament is divided, and the esophageal musculature identified above the diaphragm and cleaned of areolar and lymphatic tissue. This packet is elevated upward with the lung to, and then around the inferior pulmonary vein.

4. *Subcarinal node dissection* (Fig. 6): The lung is held anteriorly by steady, gentle traction, elevating bronchi and esophagus, and released momentarily at least every fifteen minutes. Dissection of areolar tissue off the esophagus, sparing if possible the right vagus nerve but dividing its bronchial branches, is continued upward to the level of the carina. The esophagus may then be retracted sharply posteriorly exposing the large conglomerate mass of lymphoid tissue wedged between the main bronchi. Ready and practically avascular mobilization of this nodal packet is effected by entering the plane adjacent to the posterior points of the left, or contralateral bronchial cartilages. The cartilaginous surface of the bronchus is a safe base for dissection, particularly as the nodes seldom adhere to or overlie the membranous portion of either main bronchus. Dissection is carried to the carina (Fig. 7) and sufficiently along the right main bronchus to allow its transection at the desired level. This still leaves the main mass attached by its lymphatics to the distal bronchus and lung; it remains to lift this packet off underlying pericardium. Here the avascular cleavage plane is immediately adjacent to pericardium.

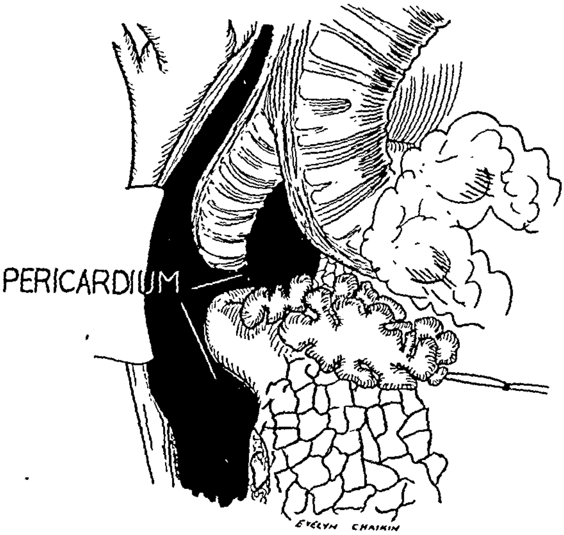

Fig. 7.—Right side: Subcarinal lymph nodes dissected onto lung from both bronchi and pericardium. Individual ligation of the bronchovascular elements can now be performed.

5. *Anterior pleuro-pericardial dissection:* All lymphatic tissue adjacent to superior vena cava and pericardium around the phrenic nerve is swept laterally, cleanly off these surfaces, and particularly between the pulmonary artery and the

superior pulmonary veins. Unless these nodes which are part of the anterior mediastinal chain are markedly enlarged by inflammatory or metastatic changes or are adherent to pericardium, intrapericardial division of the pulmonary vessels, as practiced by Allison,[1] is not done.

The node dissection, with mediastinal nodes remaining attached to the lung, is now completed, and individual division of the hilar structures can be begun (Overholt[9]).

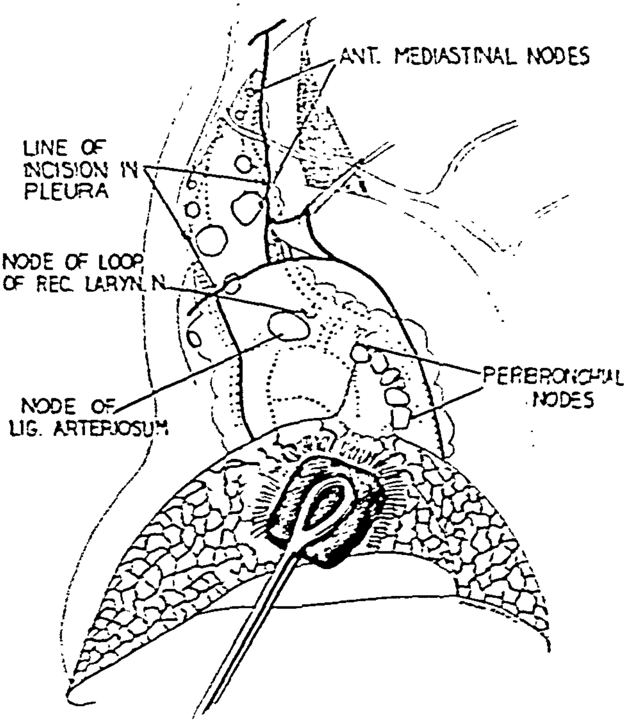

Fig. 8.—Left side: Anatomy of anterior mediastinal and subaortic lymph nodes. The pleural incision is outlined.

Alternatively, the bronchus may be divided before undertaking this subcarinal dissection (Fig. 11) or even completing the dislocation of the node of the arch of the azygos vein or clearing the superior surface of the pulmonary artery. Such an alternative is particularly applicable when a large upper lobe tumor makes access to the region of the pulmonary artery difficult, or when the nodes between the bronchus and pulmonary artery are greatly enlarged. The bronchial arteries are divided prior to bronchial transection. Traction on the distal severed stump must be gentle to avoid tearing the adjacent nodes prior to their mobilization from the opposite bronchus, pericardium, and pulmonary artery.

Left side.—

1. *Incision* (Fig. 8): This is made into the mediastinal pleura beginning at the apex of the chest and proceeding along the vagus nerve to the arch of

the aorta. Its posterior limb follows the aortic arch to the level of the bronchus and thence medial to that vessel as far as the diaphragm. Its anterior limb extends 1 to 2 cm. anterior to, and inferiorly along the course of the phrenic nerve to the inferior pulmonary ligament to join the posterior incision.

Flaps are elevated and retracted (Fig. 9). The phrenic nerve is fully liberated throughout most of its mediastinal course so that it may be retracted anteriorly and posteriorly by a Penrose drain as the dissection warrants.

2. *Anterior mediastinal dissection:* The apex of the dissection is at the undersurface of the left innominate vein at a point where it crosses the left subclavian artery. The areolar, node-bearing tissue is dissected off the vein, and several small branches must be separately identified and divided. The dissection (Fig. 10) proceeds anteriorly beneath the innominate vein as far as its entrance into the superior vena cava, dividing the highest intercostal and the thymic branches along the way. This is the anterior limit of the dissection.

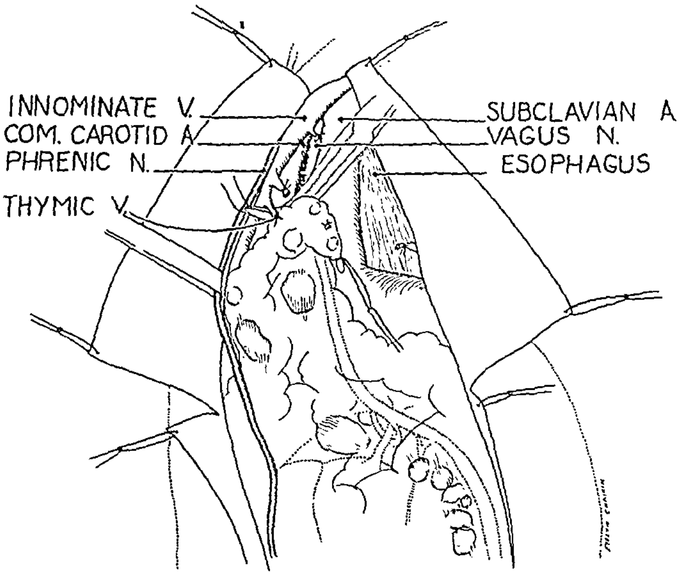

Fig. 9.—Left side: Mediastinal lymph node dissection begun along left innominate vein. Phrenic nerve is retracted anteriorly.

The posterior limit of the dissection is the subclavian artery. The packet of tissue is swept down off the subclavian, common carotid, and innominate arteries and the aortic arch. The vagus nerve is carefully cleaned and, in particular, the large node present in the interval between the common carotid and subclavian arteries is included in the specimen.

3. *Subaortic dissection* (Fig. 11): Beneath the aortic arch, the node of the ligamentum arteriosum and that of the loop of the left recurrent nerve are freed. Lying between the superior border of the pulmonary artery and the anterior surface of the left main bronchus and extending beneath the aorta is a

packet of nodes which must be dissected down toward the specimen. All of these are included with the previously dissected mediastinal packet of tissue and sewn onto the lung surface.

The pleuro-pericardial, inferior pulmonary ligament, and subcarinal dissections (Fig. 12) are similar on both sides and do not warrant special descrip-

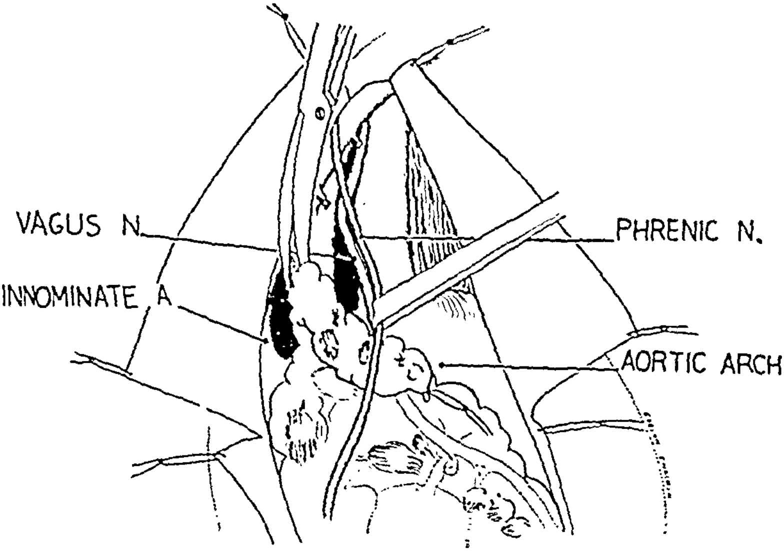

Fig. 10.—Left side: Mediastinal dissection continued. Phrenic nerve retracted posteriorly and lymph node packet passed beneath it.

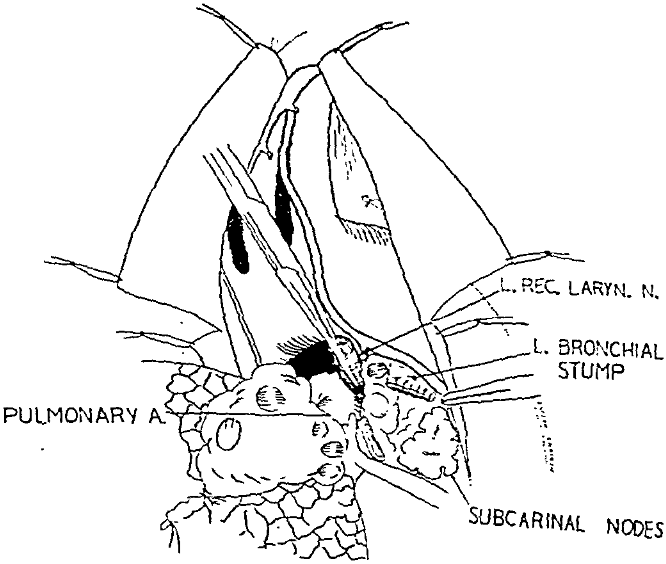

Fig. 11.—Left side: Subaortic lymph node dissection begun *after* bronchus has been divided (alternate method).

tion. The key to dissection in this area is exposure achieved by gentle traction on the lung and mobilization of the esophagus.

Closure of the superior mediastinal pleura at the conclusion of the procedure may aid in reducing subcutaneous emphysema.

MEMORIAL HOSPITAL CASES

Between January, 1949, and April, 1951, sixty pneumonectomies for cancer were performed. Twenty-one of these were simple, and thirty-nine were radical pneumonectomies (Figs. 13, 14, and 15). In the former, one postoperative death due to bronchopneumonia occurred, and in the series of radical pneumonectomies deaths were due to hemorrhage from the pulmonary artery twenty-four hours after operation, massive hemorrhage from a site in the gastrointestinal tract, undisclosed even at autopsy and hemiplegia on the fourth postoperative day. None was charged directly to the more extensive surgery.

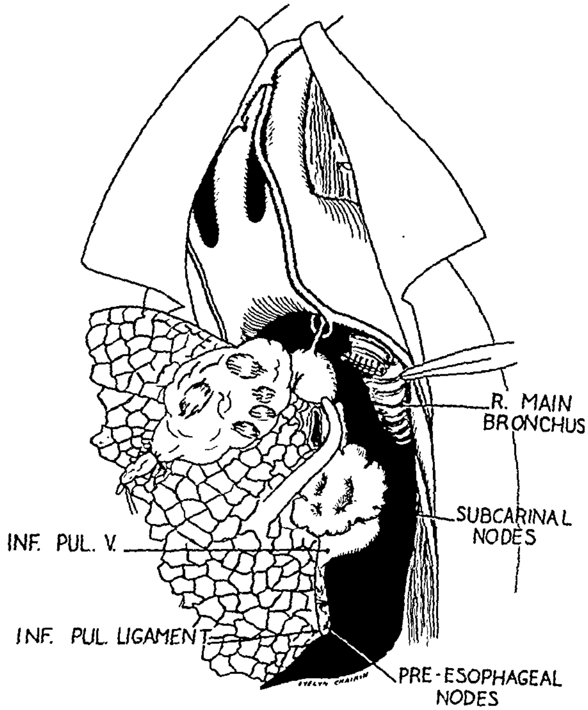

Fig. 12.—Left side: Pre-esophageal and subcarinal lymph node dissection completed. Individual ligation of vascular elements can now be performed.

In addition to the radical node dissections portions of the pericardium were resected in five instances, and of the chest wall twice. Pericardial resection accompanied simple pneumonectomy only once. The mediastinal dissection does add thirty to forty-five minutes to the operative time, but it is not difficult and, as a matter of fact, greatly aids mobilization of hilar vessels and the bronchus. About two-thirds of our cases have been actually done by resident surgeons.

The only increase in postoperative morbidity is related to the difficulty of maintaining intrapleural pressure at the desired level the first two or three days. Subcutaneous emphysema is prominent, but cardiac arrhythmias have not been more frequent, having been noted seven times following the radical and three after the simple procedure.

In the simple pneumonectomy specimens, intrapulmonic nodes contained metastases ten times, while in the radical pneumonectomy specimens metastases were found twenty-six times in these nodes and eighteen times in the mediastinal group, some of these in the apex of the dissection (Fig. 16). In no specimen were metastases found in the mediastinal nodes without also occurring in the intrapulmonic nodes. In a good many specimens the operator's impression of nodal pathology was not confirmed microscopically.

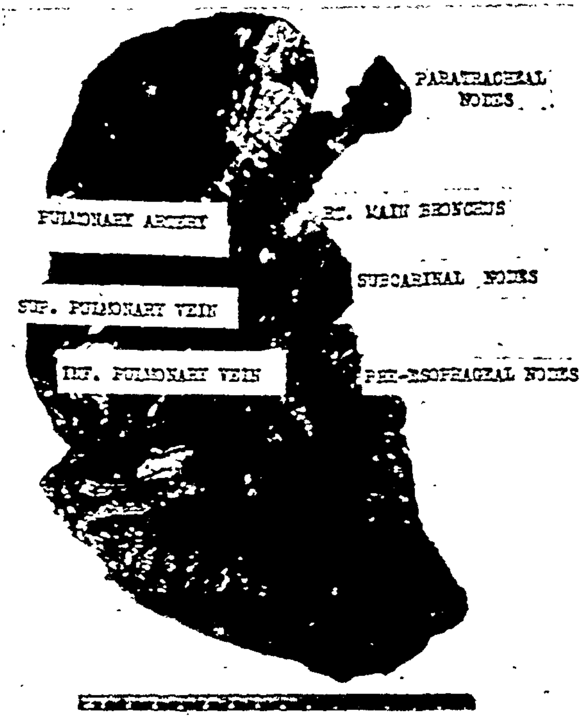

Fig. 13.—Specimen of right radical pneumonectomy, anterior view. Note block dissection.

No safe or true statistical conclusions can be drawn from so few cases and in the absence of a long follow-up period. For instance, the fact that as of April 1, 1951, nine of the twenty-one patients with simple pneumonectomies are already dead of their cancer, whereas only eleven of the thirty-nine radically operated patients have died, cannot serve as evidence in favor of radical pneumonectomy.

RATIONALE

When a primary carcinoma of the lung is centrally placed, either arising in or extending to a main-stem bronchus, the minimal procedure for total removal is a pneumonectomy. The same general rule holds true for a more peripherally placed carcinoma of the lung that invades other lobes by direct extension or by intense inflammatory reaction. Although the necessity for a complete lung removal, when feasible, is not questioned in this group, clarification is needed as to the advisability and the extent of an accompanying lymph node dissection.

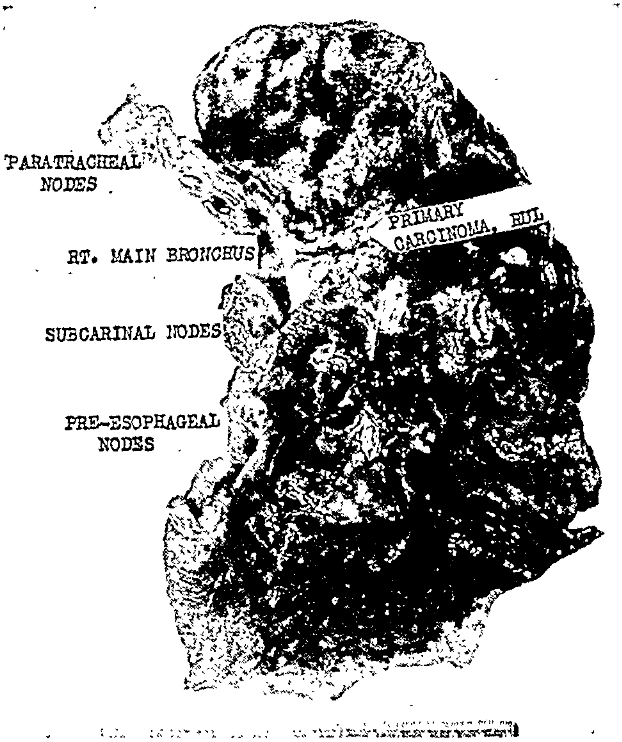

Fig. 14.—Specimen of right radical pneumonectomy, posterior view. Note block dissection and primary carcinoma.

When a primary carinoma of the lung is confined to a lobe, is peripherally placed, and is relatively small and well circumscribed, the surgeon may elect to do a less radical procedure on the grounds that he is sparing useful respiratory surface while still performing an adequate cancer operation. However, Most, as quoted by Rouviere, has said that "pulmonary lobes do not establish distinct lymphatic regions. The lymphatic tracts which run between the lobes are fed by lymph vessels from the two neighboring lobes." This seems certainly suggested by the size of the interlobar venous channels which may well be accompanied by lymphatics, and also the frequent failure of complete division of lobes anatomically. It is possible that the intrapulmonary lymph nodes which drain a particular lobe may be situated within the substance of another and, although the original lobe containing the primary focus of cancer is removed, its metastatic deposit could be left behind. If a lobectomy is elected it should be accompanied by a node dissection as radical as possible which takes into consideration those that may be involved knowing the location of the primary tumor. The dissection should include the subcarinal and the paratracheal groups, and also remove nodes about adjacent bronchi.

Small or peripheral cancers have been found to metastasize to hilar and mediastinal nodes. It is, therefore, encumbent upon us to be adequate and deal radically with these small lesions in the hope that at this one procedure, at which time the chance to cure the patient is optimum, all possible cancer and its potential areas of spread are removed.

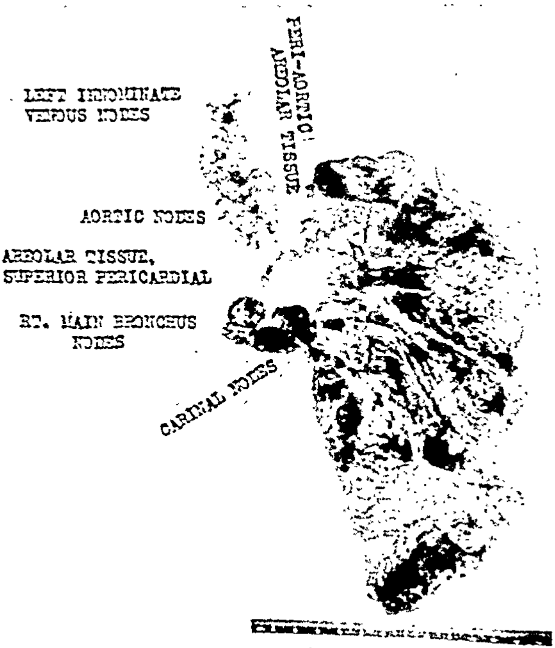
LEFT INNOMINATE VENOUS NODES
PERI-AORTIC AREOLAR TISSUE
AORTIC NODES
AREOLAR TISSUE, SUPERIOR PERICARDIAL
RT. MAIN BRONCHUS NODES
CARINAL NODES

Fig. 15.—Specimen of left radical pneumonectomy. The pre-esophageal group has not been labeled.

Although lesions of the lingula and left lower lobe are less amenable to a complete lymph node dissection for reasons already given, the excision of subcarinal nodes and those of the ipsilateral paratracheal group offers a better chance of including random metastases than if it were not done.

When a lesion is in doubt as to its being primary or metastatic, we do an aspiration biopsy through the open thorax[2] or resect a lobe and have a frozen section biopsy performed. If it then proves to have its origin in the lung, a radical pneumonectomy is completed at that time.

Koletsky[7] has noted that all of the various histologic types of carcinoma of the lung, whether centrally or peripherally located, are capable of metastasizing to the regional lymph nodes. The squamous carcinomas arising in the main bronchi seem actually to remain within the thorax, and in 65 per cent of the cases killed by local invasion and extension rather than by distant metastases. The terminal bronchiolar and adenocarcinomas comprise most of the peripheral lesions. Along with the anaplastic or oat-cell carcinomas, these are infamous for the rapidity of their vascular, extrathoracic spread, but they, too, metastasize to the regional lymph nodes with some regularity. Although the over-all prognosis for these more rapidly disseminating histologic types is poor, nevertheless when the indications for resectability exist, the radical operation should be carried out. For it is felt that the histologic type of the tumor should not be the influencing factor on the procedure to be performed, as five-year survivals in all types have been reported.

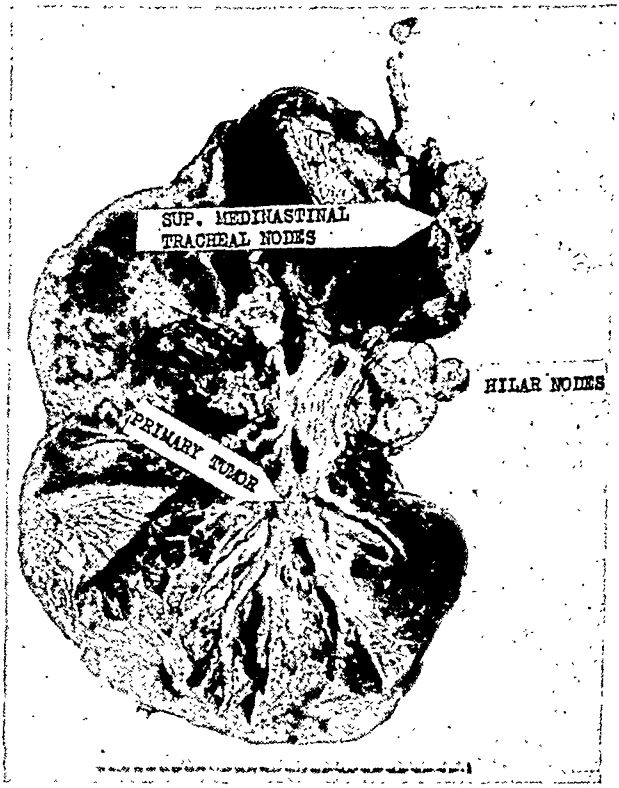

Fig. 16.—Specimen of right radical pneumonectomy showing primary carcinoma of the right lower lobe with metastases to the subcarinal ("hilar") and paratracheal ("superior mediastinal tracheal") lymph nodes.

In various communications it has been stated that the reason for leaving the hilar and mediastinal lymph nodes intact is that they felt clear of metastases.[3] Or else, one so-called representative lymph node was removed for frozen section and, when reported as negative, the others were presumed to be negative as well. Some feel that the finding of one positive lymph node warrants leaving the rest in situ, and then they do a "palliative resection." The term "palliative" used in this sense has an inherent defeatism which is at variance with the fact that there have been five-year survivals of primary lung cancer with proved metastatic hilar lymph nodes. Graham's[6] first case has gone eighteen years after the pneumonectomy specimen showed lymph nodes positive for metastases. The term also presupposes that there is certainty of metastatic lymph node involvement rather than enlargement of these structures because of other conditions.

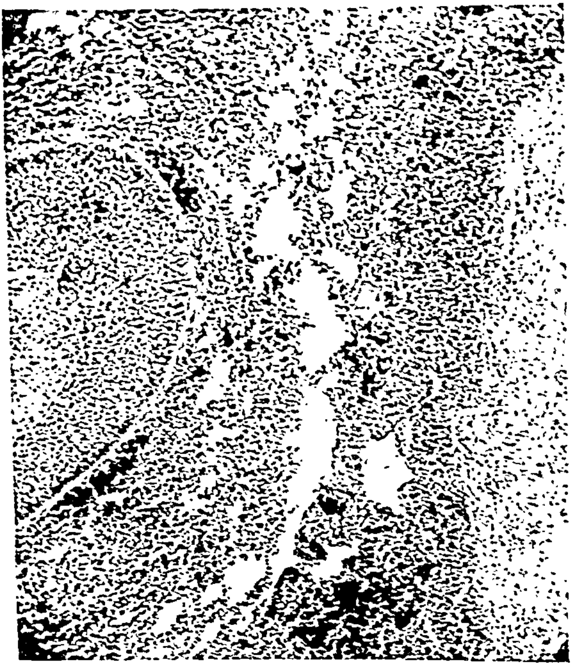

Fig. 17.—Small focus of metastatic epidermoid carcinoma of the lung in one paratracheal lymph node out of eight that were sectioned.

It is believed by the authors that it is impossible to determine the presence of metastases in a lymph node by gross observation or palpation. In fact, even the "microscope can err" in this regard. For example, Saphir and Amromin[11] re-examined axillary lymph nodes from thirty radical mastectomy specimens that had been reported uninvolved on routine examination. They found metastasis in one-third on serial sectioning of the nodes. In our own experience metastatic foci have been observed in part of one high-powered field of a lymph node section (Fig. 17). Certainly no surgeon can claim a microscopic touch of such delicacy!

If judgment as to whether or not a radical lymph node dissection is to be performed is based upon the node taken for frozen section, it must be remembered that: (a) the correct node or nodes must always be selected; (b) the microtome knife must pass through the metastatic focus; and (c) the occasional skipping of nodes in a chain makes all of them suspect and not just one alone.

The excision of all possible lymphatic areas by radical node dissection largely removes the element of conjecture about their contents. It is our belief that if there are no marked medical contraindications, a radical pneumonectomy be performed for all primary carcinomas of the lung. All procedures short of this may make a compromise with cure.

SUMMARY

1. A lung can be removed in a block dissection with most of its hilar and mediastinal lymph nodes. This procedure is called a radical pneumonectomy, and is differentiated from a simple pneumonectomy, the removal of the lung alone.
2. The procedure is based upon available knowledge of the anatomy and direction of flow of the lymphatic system of the lung.
3. From a pathologic view, this radical dissection is considered to be sound therapy for primary cancer of the lung, as the regional lymph nodes are involved frequently with metastases from both central and peripheral tumors of all histologic types; this involvement can only be determined accurately and with certainty by microscopic observation.
4. In two and one-half years, thirty-nine radical pneumonectomies have been done. Lymph nodes positive for metastases beyond the limits of the average simple pneumonectomy were found in eighteen cases.
5. It is felt that experience will clarify whether such a radical node dissection is a factor in survival for carcinoma of the lung.

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ADDENDUM

The operation as above described has been modified in several ways since its presentation.

On the right side, the procedure of excision of the right paratracheal nodes has been facilitated and expedited by sectioning, and including in this lymph node packet the vagus nerve inferior to the origin of its recurrent laryngeal branch. This requires careful identification of this branch and has not, in at least ten instances, produced any noticeable alteration in postoperative cardiac or gastrointestinal function.

On the left side, two alterations are under consideration:

1. The ductus arteriosus is ligated and divided to permit more thorough dissection in the area just beneath the arch of the aorta.
2. The first two pairs of intercostal arteries arising from the aorta are ligated and divided, permitting mobilization of the arch to allow dissection of the left paratracheal nodes that run between the arch and the trachea. This is a suggestion of Professor Pietro Valdoni of Rome, Italy.

DISCUSSION

DR. WILLIAM L. WATSON, New York.—Perhaps some of the points made by Dr. Cahan could be emphasized. He pointed out that no claim is made for originality as to the concept of an en bloc removal of a cancerous lung and its accessible lymph nodes, but he has attempted to focus attention on the fact that the operation is not only a feasible one, but is attended by an acceptable operative mortality rate of 7.7 per cent. It is to be noted also that in this series of cases two-thirds of the operations were carried out by our resident surgeons, well trained in general surgery, but some of whom have had less than three months of special training in thoracic surgery. Perhaps it should be restated that the operation of radical pneumonectomy as described by Dr. Cahan is reserved for the fair operative risk patients, and it is significant that during the period under discussion the service performed twelve lobectomies for cancer in patients who were medically unsuitable for the more radical procedure.

Not included in the statistics presented by Dr. Cahan is one case of mistaken diagnosis. A radical pneumonectomy with lymph node dissection was carried out for what proved to be an atypical form of pulmonary tuberculosis. The point of interest is that the excised lymph nodes contained active and caseating tuberculosis, leading to a question of what the future of those lymph nodes might be in relation to the possibility of contralateral postoperative spread. It might be said that in this case there was no postoperative spread. As Dr. Cahan has stated, we do not know what the ultimate salvage rate will be, but it is my hope that the radical pneumonectomy will add somewhat to the expected survival rate, but time will be required for final evaluation.

DR. J. F. HIGGINSON, Portland, Ore.—We have found this paper of Dr. Cahan's very interesting. A truly ideal cancer operation is not possible for cancer of the lung, as is the case in the breast or the neck or elsewhere. Of necessity, compromises must be made, essential structures left undisturbed, and the dissection often carried very close to the actual primary malignancy or its nodal metastases or local spread. Realizing these facts, it seemed to us that the best possible block dissection, with the least compromise, would be the most nearly ideal cancer operation in dealing with bronchiogenic carcinoma. We began planning and doing in our private services and the thoracic surgical service at the Portland, Oregon, Veterans Hospital, a block node dissection in July of 1948, and since that time we have had occasion to do forty-two pneumonectomies for bronchiogenic carcinoma. Of these, thirty-eight have had partial or complete node-bearing tissue block dissections as part of the pneumonectomy. Of the thirty-eight, ten were partial and twenty-eight complete. By partial is meant that the superior mediastinal node-bearing tissue along the trachea was left undisturbed. In all thirty-eight cases the nodes of the inferior posterior mediastinum, the homolateral hilar nodes, the subbifurcation nodes, and as many as possible of the contralateral hilar nodes were removed as part and parcel of the specimen. In most of these cases, various portions of diaphragm, mediastinal pleura and pericardium, and segments of phrenic and vagus nerves and chest wall were also removed, as is common. In some instances, portions of esophageal musculature and cuffs of the left atrial wall were removed.

We have not as yet attempted to evaluate these cacses from the standpoint of early or late results, but the pathologic findings are interesting. Of the thirty-eight cases, sixteen had nodes and twenty-two had positive nodes. If we consider only the twenty-eight complete block dissections, eleven had negative nodes and seventeen had positive nodes.

(Slide.) This is a section of a man, right side, demonstrating, as you saw in the slides of the essayists, the paratracheal superior mediastinal lymph node block; the other nodes as a complete block turned up from the contralateral bronchus, sub-bifurcation area, and homolateral bronchus are included.

(Slide.) This is another right-sided lesion without a card to make the nodes stand out; the nodes are tossed up over the top of the specimen. You can see the other nodes gathered together in a smooth continuous clump next to the bronchus.

(Slide.) Another right-sided lesion, again demonstrating that this group of node-bearing tissue can be removed en bloc with the lung.

(Slide.) This is a left-sided lesion demonstrating pericardium and the inferior pulmonary vein; this vein was totally occcluded by carcinoma and this was bulging into the left atrial lumen as a polypoid thumblike mass. The nodes in this man's specimen were negative.

DR. JOHN M. CHAMBERLAIN, New York.—I did not intend to discuss this paper, but I find it extremely interesting and worth while, and so I rise mainly to raise one question and to invite discussion from the authors.

Since Allison and Bloch first called this type of resection to our attention, we have been trying a more radical procedure, but have wondered whether we should leave the lung in and do the radical dissection first, or whether we should remove the lung and do the radical mediastinal dissection later. If the mediastinal dissection is done first, extensive manipulation of the lung is necessary. Should the vein be invaded, this varied dislocation of the lung could conceivably dislodge small emboli into the systemic circulation. All of us, I am sure, occasionally have found the veins invaded at operation and have worried, therefore, about the dissemination of such emboli. Perhaps veins within the parenchyma are always invaded. On the other hand, Drinker has shown that ligation of the veins tremendously increases the peribronchial lymph flow. We have therefore determined operability with as little manipulation of the lung as possible, and then proceeded to clamp and divide the bronchus immediately, ligating the vessels next. After removal of the specimen, the radical dissection is accomplished.

Today, Dr. Cahan pointed out that they begin their radical dissection in the mediastinum. So I raise this point: I wonder which is the more hazardous as a result of the operation—dissemination via the veins through excessive manipulation of the lungs, or via the lymphatics, the most distant of which are interrupted first, as outlined by the authors.

DR. ADRIAN LAMBERT, JR., New York.—I, too, should like to compliment Dr. Cahan on his fine presentation. I wish that the actual procedure were as easy as some of these pictures portray. In our experience it is much more difficult to dissect out the mediastinum than these drawings would have us believe.

This procedure is one that should be performed in cases in which the patient can stand the operation. At Bellevue we have employed radical mediastinal excision when this is the case, and have been doing it more and more in routine removal of carcinoma of the lung. There are one or two points I should like to make. First, the value of this procedure will depend on the relation of five-year survival to the type of cases selected for radical dissection. In cases in which we cannot remove the entire lung for functional reasons, where the patient cannot stand the entire dissection—and there are cases we encounter in which that is so—it is wise to bear in mind one or two points which may be of value. One is that carcinoma of the upper lobe may metastasize to the carinal node, so that if the mediastinal extension is to be removed, that node should be removed too. Carcinoma of the lower lobe more frequently goes to the lower nodes, so that in doing a partial resection that part should be removed first. Carcinoma of the upper lobe, in my experience, does not go to the lower nodes down toward the diaphragm without first extending upward, so that in doing a partial dissection, this is worth bearing in mind.

The third point I should like to mention is the presence of a structure which is not emphasized in the anatomy books. From studies of bodies that I have dissected and patients encountered at the operating table, there seems to be a fibrous membrane situated longitudinally posterior to the trachea extending down to the esophagus. This seems to be a constant finding and appears to prevent extension in the mediastinum, in this area at least, from one side to the other.

One note about complications: These patients are prone to develop mediastinal emphysema which should be controlled with negative intrathoracic pressure or suction. In addition to having difficulty coughing, and a large amount of secretions, they also seem to develop edema of the opposite lung, which brings up the question of whether the lymph drainage to the opposite lung may be impaired. I do not feel that it has been determined how compatible with life it is if you remove all the peribronchial nodes on the other side.

I feel that this procedure should be done more frequently, and I want to compliment the men from Memorial on directing attention to its more routine use.

DR. CAHAN (closing).—I would like to thank Dr. Watson, Dr. Higginson, Dr. Chamberlain, and Dr. Lambert for their discussions, and to take up briefly some of the points they have raised.

Dr. Higginson mentioned the point that Dr. Churchill quoted last year about there being a morass of lymphatics in the mediastinal area. To be sure that is so, but we like to emphasize that these can be removed for the most part en bloc, and although we do probably make certain compromises with certain lymphatics. I feel that this is certainly true also in operations performed on the breast in which there are parasternal lymph nodes as possible foci of metastases; also in the stomach and colon metastatic spread may well extend beyond the standard limits of resection.

Dr. Chamberlain's question of sequence of operation—doing the pulmonary vein or the bronchus first—is something about which we could talk at great length. We apply the principle of cancer surgery and take the distalmost point of possible lymphatic spread first, working back toward the primary itself. The question of avoiding venous emboli is a good point, and I think we should consider it carefully.

With Dr. Lambert's statement about the simplification of our pictures I agree wholeheartedly. But I am sure that the people in the back row, if all the ties and fibrous tissue were inserted into the picture, would be indeed confused. I agree that there are points in the dissection that are more difficult than others, but these can be surmounted with patience. But basically, when you get that lymphatic packet, for example in the paratracheal or retrocaval areas on the right side, it does seem to come down with relative ease and well ccontained, particularly if you hug close to the major vessels and to the trachea. We have not seen the edema of the opposite lung noted by Dr. Lambert.

PLEUROPULMONARY RESECTION WITH HEMIDIAPHRAGMECTOMY

Preliminary Report

Bert H. Cotton, M.D., and Joao R. F. Penido, M.D. (by invitation)
Beverly Hills, Calif.

THE present paper consists of a discussion of indications, complications, technical problems, and results in a series of twenty-eight cases of coexisting pulmonary and pleural disease treated by surgical methods. Each of the twenty-eight patients received pulmonary resection combined with pleurectomy. In eleven instances, subtotal or total hemidiaphragmectomy was performed as an adjunct to pleuropulmonary resection. The results obtained are encouraging.

PLEUROPULMONARY RESECTION

For many years chest surgeons have performed pneumonectomy with pleurectomy merely for reasons of technical convenience. Pleuropulmonary resection has been employed successfully in the management of tuberculosis with complicating empyema. However, little use has been made of this combined procedure in the treatment of malignancy. Our experience indicates that pleuropulmonary resection may prove lifesaving in cases of bronchial carcinoma heretofore considered hopeless and will increase the present salvage rate in routine resectable cases.

HEMIDIAPHRAGMECTOMY

The value of subtotal or total hemidiaphragmectomy as an adjunct to the treatment of thoracic malignancy also has not been fully appreciated. If the malignant process involves the diaphragmatic pleura, either by direct extension or by implants, we have found a hemidiaphragmectomy combined with pleuropneumonectomy makes the operation more definitive for removal of all neoplastic implantations. In our opinion, previous reports in the literature have placed too great importance on preserving the continuity of the diaphragm. Our experience is that a subtotal or total hemidiaphragmectomy can be performed without detrimental effects on the patient.

The fate of the diaphragm depends upon the stage of pleural involvement. If no involvement has occurred because of a localized pocket of pleural fluid, the diaphragm is allowed to remain intact. Pleuropulmonary resection with subtotal hemidiaphragmectomy is performed when segmental involvement of the diaphragm is present. If the entire diaphragm is involved, complete hemidiaphragmectomy is the treatment of choice.

In a large percentage of our patients we have found it impossible to remove the pleura from the diaphragm without causing rupture into the pleural space. The dissection thus is facilitated by removal of the diaphragm.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

One complication of hemidiaphragmectomy is the pronounced shortness of breath. We have surmised that this manifestation is due to loss of the central attachment of the contralateral leaf of the diaphragm. The shortness of breath remains for about seven to ten days postoperatively, after which the mediastinum is stabilized. Apparently the thick fibrinous membrane which is formed over the peritoneum acts as a buffer as well as a solid attachment for the remaining leaf of the diaphragm.

In cases of tuberculosis with empyema, the danger of abdominal infection following pleurodiaphragmectomy may be avoided by preserving the peritoneum; this structure provides a base upon which a fibrinous membrane rapidly forms. Recurrent empyema or bronchopleural fistula will be late postoperative complications, allowing an interval during which a heavy fibrinous wall forms between the abdominal and thoracic cavities.

ANALYSIS OF AUTHORS' CASES

Tables I, II, and III show the types of cases included in the present study and the treatment employed in each type. The twenty-eight instances may be divided into two major classifications, that of malignancy and that of infection. Surgical treatment in cases of the infective type is less radical than in malignancy, where only wide excision can improve the prognosis.

TABLE I

1. Primary carcinoma of the lung with associated pleural effusion and malignant cells	8
2. Diffuse primary neoplasm of the pleura	2
3. Pulmonary tuberculosis with tuberculous empyema	9
4. Pulmonary fungus cavities with rupture and empyema	8
5. Bronchiectasis with empyema	1

1. *Primary Carcinoma of the Lung With Associated Pleural Effusion and Malignant Cells* (8 cases).—When malignancy of the lung has involved the parietal or visceral pleura by direct extension or implantations with or without pleural effusion, pleuropulmonary resection with pleurodiaphragmectomy often will decrease morbidity and mortality. In such instances this combination of operations may be considered a routine procedure unless a protective cushion of lung is present between the lesion and the pleura.

Five of the eight patients are alive and well after postoperative periods of two years, one year, nine months, six months, and four months, respectively. Two patients died with metastasis less than a year after operation. One 72-year-old white woman died of coronary occlusion twenty-four hours postoperatively.

2. *Diffuse Primary Neoplasm of the Pleura* (2 cases).—This neoplasm, known as diffuse mesothelioma or endothelioma of the pleura, involves the entire pleura and usually ensheathes the lung in tumor tissue of the diffuse type. The two such cases in the present series were treated by pneumonectomy with hemidiaphragmectomy. One patient presented erosion through the

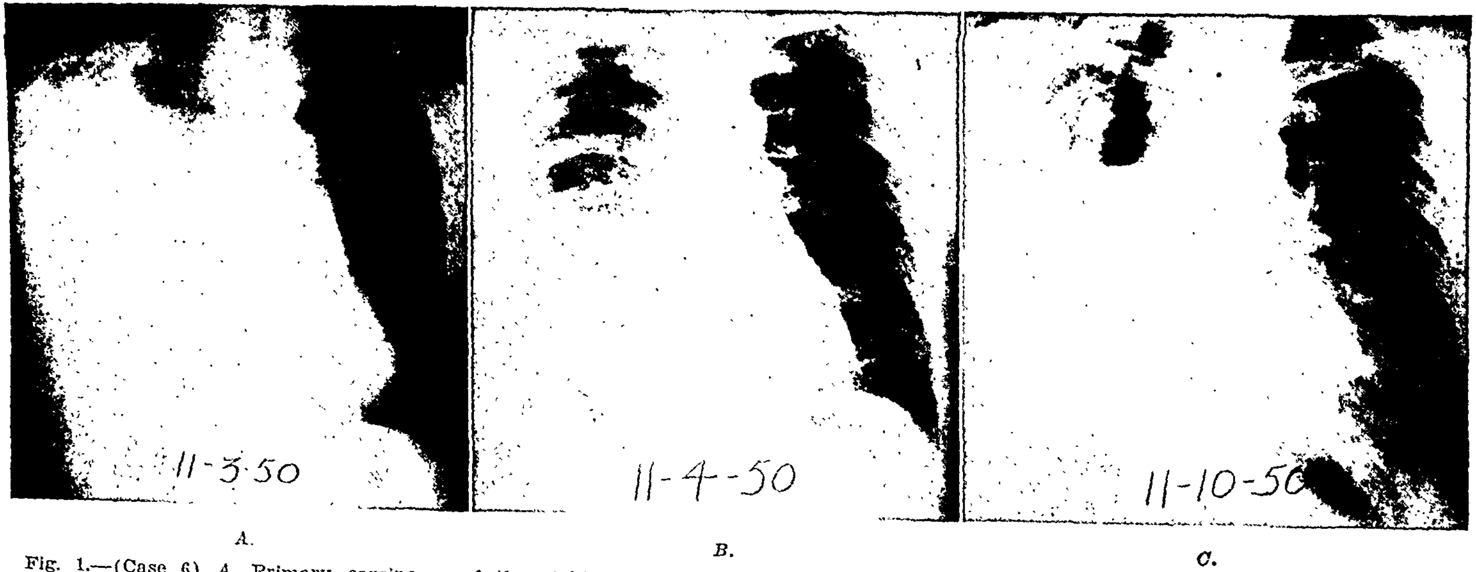
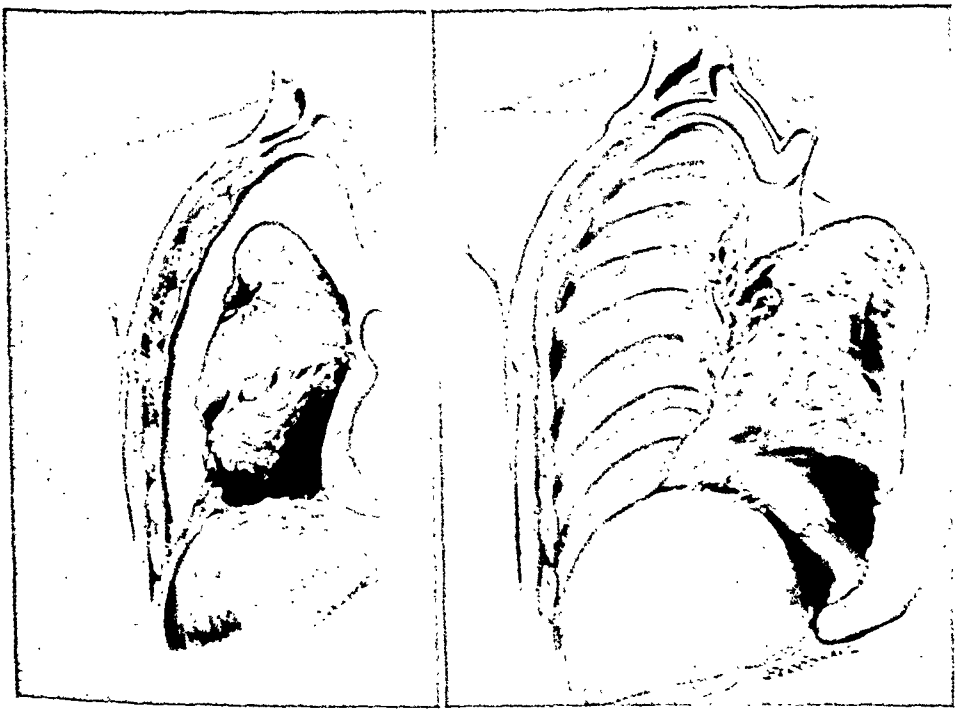


Fig. 1.—(Case 6) *A*, Primary carcinoma of the right lung with associated pleural effusion and malignant cells; *B*, removal of fluid with demonstration of the small lesion in the midlung area; *C*, second postoperative day, no abdominal viscera in right hemithorax.

diaphragm with involvement of the left adrenal gland and left kidney; these organs also were resected. The patient progressed well for fourteen months postoperatively, then died of cerebral vessel rupture. The second patient lived seven months after operation and died of abdominal metastasis.

3. *Pulmonary Tuberculosis With Empyema* (9 cases).—In our experience surgical treatment of pulmonary tuberculosis with positive sputum in combination with tuberculous empyema has been discouraging. Our disappointment may be attributed to: (1) a high mortality rate; (2) the *long, complicated surgical regimen* necessitating skeletal deformity; and (3) failure to control pulmonary disease due to the extrapulmonic heavy fibroblastic membrane over the pleura.



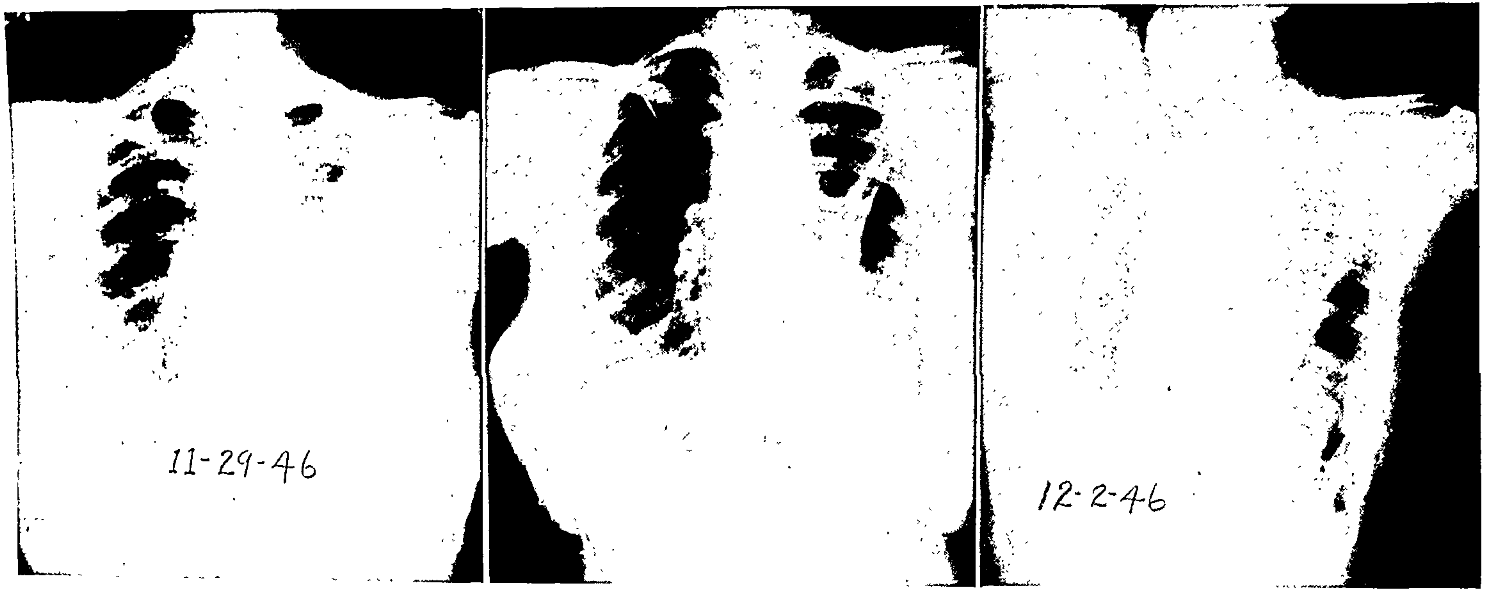
A. *B.*

Fig. 2.—(Case 6) *A*, The appearance of the lesion at the operating table; *B*, pleuropneumonectomy with hemidiaphragmectomy.

Thoracoplasty treatment is directed primarily toward cure of the empyema. It is hoped that the pulmonary disease also is cured. Extrapleural pulmonary resection is definitive for both pulmonary and pleural tuberculosis.

All of the nine patients recovered. One patient developed a late bronchopleural fistula, treated successfully with sodium hydroxide cautery, drainage, and thoracoplasty.

4. *Fungus Disease of the Lung With Ruptured Cavity and Empyema* (8 cases).—Pleuropulmonary resection is proving of value in the management of fungus diseases of the lung and pleura. In our series of sixty cases of pulmonary fungus disease treated by surgical means, eight patients exhibited



A. B. C.

Fig. 3.—(Case 10) *A*, Diffuse primary neoplasm of the left pleura with pleural effusion and malignant cells; *B*, removal of the fluid with diagnostic pneumothorax, neoplasm demonstrated; *C*, oblique view showing the multiple tumor growths of the pleura.

coexisting pleural and pulmonary involvement. Each of these eight patients received pleuropulmonary resection, recovered with no complications, and returned to normal life.

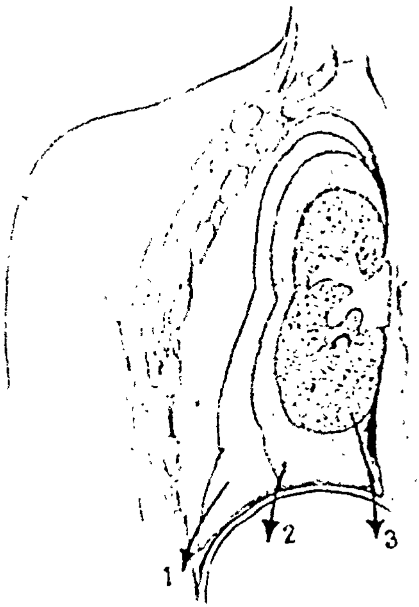


Fig. 4.—The fate of the diaphragm depends upon the pleurodiaphragmatic involvement. 1. Hemidiaphragm remains intact. 2. Selected [illegible] pleurectomy. 3. Hemidiaphragmectomy.

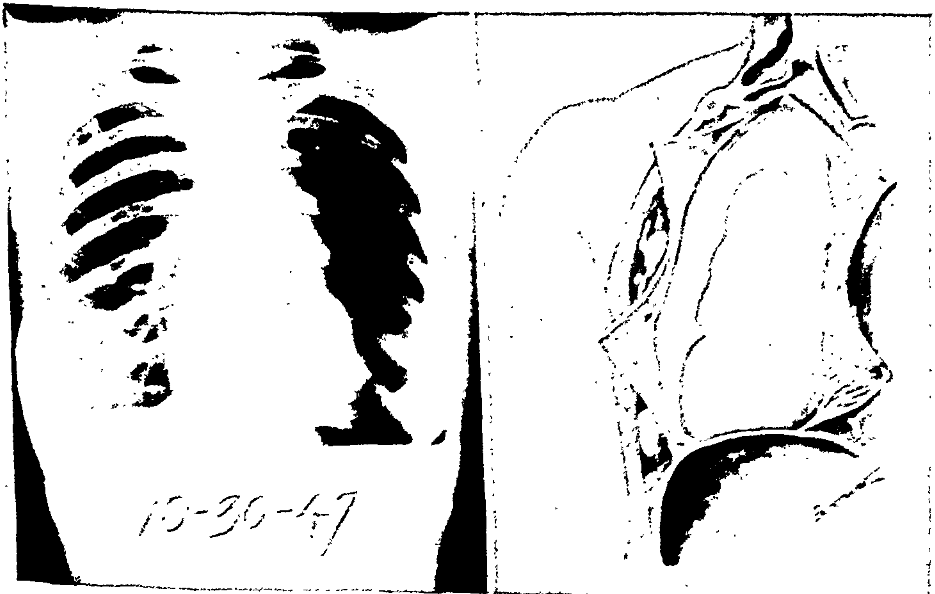
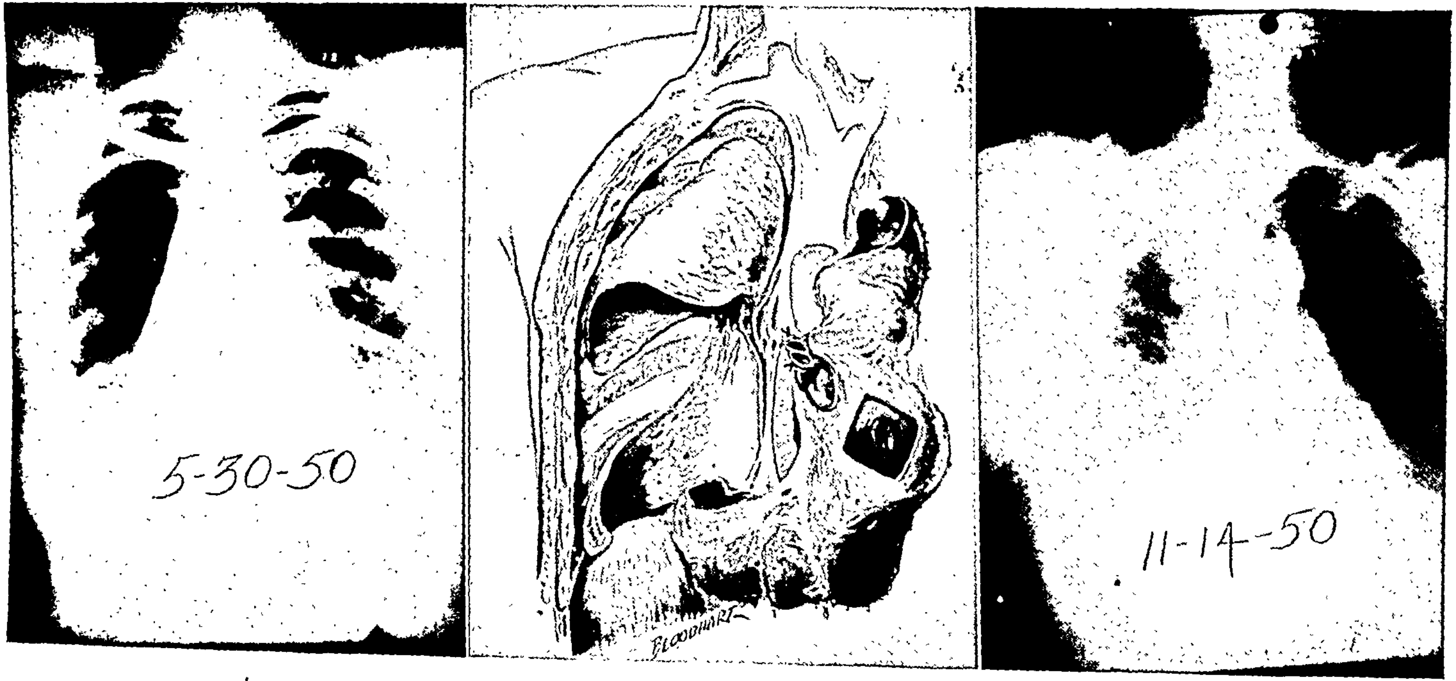


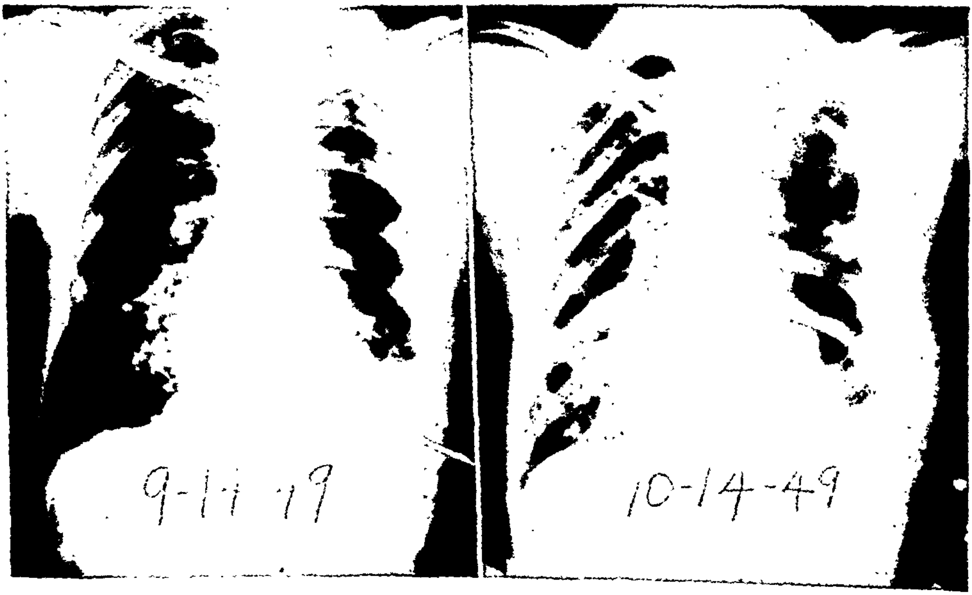
Fig. 5.—Case 11. A, [illegible] of the left lung with tuberculous empyema. B, appearance of the [illegible] and pleura at the operating table. Pleuropneumonectomy was performed.



A. B. C.

Fig. 6.—(Case 19) *A*, Pulmonary tuberculosis and coccidioidomycosis of the right middle lobe, tuberculous and coccidioidal empyema was also present; *B*, right pleurolobectomy of the middle lobe which also included decortication of the right upper and lower lobes; *C*, postoperative films with right upper and lower lobes reexpanded.

5. *Bronchiectasis With Bronchopleural Fistula and Empyema* (1 case).—Profound disagreeable symptoms, periodic incapacitation, and early death has been the fate of patients with bronchiectasis complicated by bronchopleural fistula and empyema. The one such patient in our series developed a lung abscess which ruptured, resulting in suppurative empyema with bronchopleural fistula. Closed catheter drainage was present when the patient was first seen by us. Left pleuropneumonectomy was performed after which the patient progressed satisfactorily until a bronchopleural fistula developed on the fourteenth postoperative day. This was treated successfully by drainage, sodium hydroxide, cauterization, and thoracoplasty. The patient has now returned to work.



A. B.

Fig. 7.—(Case 28) *A*, Bronchiectasis of the left lung with bronchopleural fistula and empyema; *B*, postoperative film one week following left pleuropneumonectomy.

TABLE II. SURGICAL PROCEDURES EMPLOYED

1. Pleuropneumonectomy	5
2. Pleuropneumonectomy and hemidiaphragmectomy	5
3. Pleuropneumonectomy and segmental diaphragmectomy	6
4. Pleurolobectomy	6
5. Segmental pleurolobectomy	6
Total	28

SURGICAL TECHNIQUE

Through the bed of the resected rib, an extrapleural plane of dissection is found and enlarged sufficiently to permit the use of a rib spreader. A small incision is made in the parietal pleura, and the intrapleural contents explored. The fluid content is aspirated; this precaution prevents widespread contam-

TABLE III. PRIMARY CARCINOMA OF THE LUNG WITH ASSOCIATED PLEURAL EFFUSION AND MALIGNANT CELLS

CASE	AGE (YR.)	SEX	RACE	DIAGNOSIS CONFIRMED BY	EXTENT OF INVOLVEMENT	CHARACTER OF FLUID	OPERATIVE PROCEDURE	COMPLICATIONS	PRESENT CONDITION
#1 W. S.	57	M	W	Malignant cells in pleural space	Total pleura involved	Amber	Left pleuropneumonectomy with hemidiaphrectomy and resection of chest wall	P.O. shortness of breath	Living P.O. 2 yr.
#2 M. M.	69	F	W	Malignant cells in pleural space	Total pleura and one-half of left diaphragm involved	Amber	Left pleuropneumonectomy with subtotal hemidiaphragmectomy and resection of chest wall	P.O. shortness of breath	Living P.O. 1 yr.
#3 I. C.	51	M	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Left pleuropneumonectomy with subtotal hemidiaphragmectomy	P.O. shortness of breath	Living P.O. 9 mo.
#4 F. D.	67	W	M	Malignant cells in pleural space	Total pleura, no diaphragm involved	Amber	Right pleuropneumonectomy with chest wall resection	None	Living P.O. 4 mo.
#5 E. O.	53	M	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Right pleuropneumonectomy with subtotal hemidiaphragmectomy	None	Living P.O. 4 mo.
#6 A. B.	66	M	W	Malignant cells in pleural space	Total pleura and all of diaphragm involved	Amber with blood	Right pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 4 mo. extension of disease
#7 J. P.	61	M	W	Malignant cells in pleural space	Total pleura and all hemidiaphragm involved	Amber	Left pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 8 mo. extension of disease

#8 N. L.	72	F	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Right pleuropneumonectomy with subtotal *hemidiaphragmectomy*	Coronary occlusion	Dead P.O. 21 hr.
					Diffuse Primary Neoplasm of the Pleura				
#9 F. R.	11	M	W	Malignant cells in pleural space	Total pleura and all of hemidiaphragm involved	Amber	Left pleuropneumonectomy with hemidiaphrectomy and left *nephrectomy* and adrenalectomy	P.O. shortness of breath	Dead P.O. 11 mo.
#10 B. C.	32	F	W	Malignant cells in pleural space	Total pleura and all of hemidiaphragm involved	Amber	Right pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 7 mo.
					Pulmonary Tuberculosis With Tuberculous Empyema				
#11 G. H.	20	M	W	Sputum and pleural fluid positive for tuberculosis	*Subtotal pleura*, no diaphragm involvement	Purulent	Right pleuropneumonectomy	None	Alive and well
#12 D. E.	37	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura, no diaphragm involvement	Purulent	Right pleuropneumonectomy	None	Alive and well
#13 R. T.	40	M	W	Sputum and pleural fluid positive *for tuberculosis*	Subtotal pleura and one-third of diaphragm involved	Purulent	Right pleuropneumonectomy and *subtotal* hemidiaphragmectomy	Bronchial fistula developed 2 mo. P.O.	Alive and well
#14 L. A.	28	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm *involvement*	Amber cloudy	Left upper pleurolobectomy. Decortication of lower lobe	None	Alive and well

TABLE III—CONT'D

CASE	AGE (YR.)	SEX	RACE	DIAGNOSIS CONFIRMED BY	EXTENT OF INVOLVEMENT	CHARACTER OF FLUID	OPERATIVE PROCEDURE	COMPLICATIONS	PRESENT CONDITION
#15 W. E.	53	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm involvement	Purulent	Left pleuropneumonectomy	None	Alive and well
#16 R. B.	48	F	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and one-fourth diaphragm involved	Purulent	Right upper pleurolobectomy. Decortication of lower lobes	None	Alive and well
#17 I. U.	45	F	W	Sputum positive for tuberculosis and coccidioidomycosis. Pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm involvement	Purulent	Right upper pleurolobectomy. Decortication	None	Alive and well
#18 A. B.	39	F	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura involved	Purulent	Right middle pleurolobectomy	Drainage from previous wound for 4 mo.	Alive and well
#19 E. G.	50	F	W	Sputum and pleural fluid positive for tuberculosis and coccidioidomycosis	Subtotal pleura involved	Purulent	Right middle pleurolobectomy	None	Alive and well
				Pulmonary Fungus Cavities With Rupture and Empyema					
#20 G. W.	49	M	W	Pleural fluid positive for coccidioidomycosis	Subtotal pleural involvement	Purulent	Right lower pleurolobectomy	None	Alive and well

#21 R. L.	30	F	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right lower pleurolobectomy	None	Alive and well
#22 L. M.	30	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left upper segmental pleurolobectomy	None	Alive and well
#23 D. B.	41	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right upper segmental pleurolobectomy	None	Alive and well
#24 J. T.	19	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left upper segmental pleurolobectomy	None	Alive and well
#25 L. H.	19	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left lower segmental pleurolobectomy	None	Alive and well
#26 C. B.	44	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right upper segmental pleurolobectomy	None	Alive and well
#27 H. R.	43	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left lower segmental pleurolobectomy	None	Alive and well
					Bronchiectasis Wtih Empyema				
#28 H. G.	40	M	W	Mixed infection	Total pleural involvement	Purulent	Left pleuropneumonectomy	Bronchial fistula 10 days P.O.	Alive and well. Fistula healed

ination if the pleura is inadvertently opened during the dissection. If pulmonary resection is decided upon, the pleural wound is closed with a running suture, inverting the edges. The extrapleural plane is then dissected around the entire chest cavity, down to the diaphragm, over the apex, and down the mediastinal surface to the hilar structures. In instances of involvement of the diaphragm, the insertions are dissected away from the chest wall by blunt dissection; it is sometimes easier to start with the lateral insertions. The peritoneum is located and left intact while the diaphragm is dissected away. The medial attachment is divided beyond the pleura in the mediastinum. On the right side, the medial attachment occasionally is freed from the inferior vena cava only with difficulty. In malignant cases, if there is involvement around the hilum, or if the distal portions of the vessels seem to be involved, the pericardium is entered and the vessels secured inside the pericardium. If the pleura cannot be separated from the pericardium, this structure is removed. When there is only segmental involvement of the diaphragm, a small segment of the muscle is resected with the diaphragmatic pleura. When a large portion of the diaphragm is involved, either by multiple pleural implants or by direct extension, the whole hemidiaphragm should be resected. In four of our patients postoperative chest wall defects have been repaired with stainless steel wire mesh: two of the defects were due to invasion of the chest wall by primary carcinoma of the lung; the remaining two were of localized malignant mesotheliomas.

SUMMARY AND CONCLUSIONS

1. The surgical treatment of a series of twenty-eight cases of serious coexisting pulmonary and pleural disease is described. Pleuropulmonary resection was done in each instance. Subtotal or total hemidiaphragmectomy was performed as adjunctive treatment in eleven instances. Only one postoperative death was encountered; the remaining patients tolerated the surgical regimen.

2. The authors' experience suggests that pleuropulmonary resection and hemidiaphragmectomy may prove of value in the treatment of primary carcinoma of the lung and pleura.

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DISCUSSION

DR. W. EMORY BURNETT, Philadelphia.—I want to congratulate the essayists on the presentation of an interesting and rather intriguing subject. I would like to discuss the malignant category only, and state some of our experiences with this.

About three years ago we decided to test the verity of the contraindication of pleural invasion to pneumonectomy, in the hope that this extension might be the only site of secondary invasion. The first patient on whom we intended to try this had been discharged during my absence at this meeting, I believe, to a cancer hospital. When I returned and tried to get him back in, we found he had died the day before of atelectasis of the opposite lung, but at post-mortem there was no invasion whatever of the mediastinal glands. The only secondary spread was that of the pleura, so that we were encouraged to hunt out others.

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I am pleased to note that there have been some patients in this series reported today who have gone longer, and we understand from four months to two years. I would like to know how many were for two years when the essayist returns.

DR. LOUIS R. DAVIDSON, New York.—I am sure you will agree with me when I say such a wholesome and substantial meal as was served us by Dr. Cotton needs no dessert. Because of this fact, it is with great reluctance that I discuss his paper.

About the time that Churchill and Klopstock reported upon lobectomy in pulmonary tuberculosis, it became apparent to thoracic surgeons that many upper lobes could not be resected from the chest wall, unless one was willing to risk spillage into and infection of the intrapleural space. It was natural, therefore, to seek another avenue of approach in those so-called frozen lobes or lungs. For reasons which must be quite clear, surgeons who had previous training in pathology were quick to use, in part at least, what is known in the autopsy room as the Rokitansky technique—that is what is now called extrapleural dissection or mobilization.

At Sea View Hospital, about 400 patients have been submitted to excisional surgery. Of this number, about eighty or more have had extrapleural resectional surgery. I shall confine my discussion to twenty-nine cases of mixed infection tuberculous empyema occurring in a series of 200 consecutive cases of excisional surgery performed between March, 1946, and February, 1949.

Prior to 1946, various surgeons when reporting on mixed infection tuberculous empyema indicated mortality figures in the neighborhood of 50 per cent. Dr. Cotton's figure, you will note, is lower than this. At the time pneumonectomy and pleurectomy were used at Sea View Hospital, in this group of twenty-nine cases the ultimate mortality rate was 10.3 per cent; three patints having died.

Technically, pneumonectomy and pleurectomy are more difficult for the surgeon and immediately more serious for the patient when compared with the previous methods of thoracotomy, multiple-stage thoracoplasty, and Schede. However, with pneumonectomy and pleurectomy, the end results are far superior.

ination if the pleura is inadvertently opened during the dissection. If pulmonary resection is decided upon, the pleural wound is closed with a running suture, inverting the edges. The extrapleural plane is then dissected around the entire chest cavity, down to the diaphragm, over the apex, and down the mediastinal surface to the hilar structures. In instances of involvement of the diaphragm, the insertions are dissected away from the chest wall by blunt dissection; it is sometimes easier to start with the lateral insertions. The peritoneum is located and left intact while the diaphragm is dissected away. The medial attachment is divided beyond the pleura in the mediastinum. On the right side, the medial attachment occasionally is freed from the inferior vena cava only with difficulty. In malignant cases, if there is involvement around the hilum, or if the distal portions of the vessels seem to be involved, the pericardium is entered and the vessels secured inside the pericardium. If the pleura cannot be separated from the pericardium, this structure is removed. When there is only segmental involvement of the diaphragm, a small segment of the muscle is resected with the diaphragmatic pleura. When a large portion of the diaphragm is involved, either by multiple pleural implants or by direct extension, the whole hemidiaphragm should be resected. In four of our patients postoperative chest wall defects have been repaired with stainless steel wire mesh: two of the defects were due to invasion of the chest wall by primary carcinoma of the lung; the remaining two were of localized malignant mesotheliomas.

SUMMARY AND CONCLUSIONS

1. The surgical treatment of a series of twenty-eight cases of serious coexisting pulmonary and pleural disease is described. Pleuropulmonary resection was done in each instance. Subtotal or total hemidiaphragmectomy was performed as adjunctive treatment in eleven instances. Only one postoperative death was encountered; the remaining patients tolerated the surgical regimen.

2. The authors' experience suggests that pleuropulmonary resection and hemidiaphragmectomy may prove of value in the treatment of primary carcinoma of the lung and pleura.

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DISCUSSION

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Of the twenty-nine cases of mixed infection tuberculous empyema, ten had bronchopleural fistula demonstrated preoperatively. Of the remaining nineteen cases, we have assumed from a pathologic as well as from a clinical viewpoint that all empyemas of this type at one time had had bronchopleural fistula.

Results.—

A. Twenty-four, or 82.7 per cent, are living and well with negative sputum.
B. Two, or 7.0 per cent, are living and well, but the sputum is positive.
C. Three, or 10.3 per cent, died.

Of course, pneumonectomy and pleurectomy prolong the operating time and invite a greater loss of blood. However, in another series, we have found that in all types of cases having pneumonectomy and pleurectomy the complications are more frequently noted upon the right side. These difficulties in part are: involvement of the superior vena cava, esophageal fistula, trauma to the diaphragm, etc.

When indications are followed, pneumonectomy and pleurectomy should become a well-established procedure yielding exceptional results.

I enjoyed Dr. Cotton's presentation very much.

DR. DWIGHT E. HARKEN, Boston.—I should like to confine myself to this section of the paper that involves what we, in our Clinic, would regard as meddlesome surgery for obviously hopelessly inoperable carcinoma of the lung. Our tendency has been to do less and less rather than more and more; in other words, to try to make a cleaner, clearer distinction between inoperability and operability and to work toward a more specific procedure consistent with a cancer operation. We have explored the neck and removed lymph nodes routinely in an effort to establish inoperability before proceeding with surgery. This neck dissection is also useful as a diagnostic maneuver in some instances where a diagnosis of cancer cannot otherwise be made. In such a situation it not only establishes the diagnosis but the inoperability thereof. We have, I believe, by this dissection of the neck spared some 50 per cent of our patients surgery that was otherwise nonproductive. We can be guilty of errors of commission as well as errors of omission. The excision of malignant tumors is not always ''palliative.''

DR. IRVING A. SAROT, New York.—The surgical principle of complete excision has been achieved by pleuropneumonectomy in cases of combined pleuropulmonary disease in tuberculosis, nontuberculous suppuration, and malignancy with secondary suppuration and empyema. Dr. Cotton is to be congratulated on extending the applicability of the procedure by demonstrating the feasibility of adjunctive hemidiaphragmectomy.

Inasmuch as total pneumonectomy and pleurectomy have been discussed here and elsewhere, I should like to confine my discussion to the treatment of pleuropulmonary disease in which only part of the lung is involved; that is, to the development of subtotal resection of the lung with pleurectomy and excision of chronic empyema.

(Slide.) I have found that even the largest empyemas, which may extend over the diaphragm and pericardium, the mediastinal pleura about the hilus, and the subjacent visceral pleura are not usually involved to any great degree by the inflammatory process, as shown in these specimens. This knowledge has been utilized in the development of the extrapleural mediastinal approach which facilitates pleuropulmonary resection.

(Slide.) This sketch illustrates the extrapleural approach to the uninvolved mediastinal aspect, or in less extensive localized empyemas, to the less thickened parietal pleura a short distance away from the empyema. When the thin, relatively uninvolved mediastinal pleura is incised, the visceral pleura is exposed and the proper cleavage plane is entered in which to separate the lung from the overlying thickened parietal pleura and the visceral wall of the empyema. In this manner a lobe of the whole lung may be of the empyema. In this manner a lobe of the whole lung may be freed (decorticated) from the empyema, palpated, and inflated to add information to the preoperative clinical and radiologic evidence upon which to decide whether a segment, a lobe, or the entire lung must be resected with the empyema. While still working in a clean field, one may then proceed with segmental resection, lobectomy, or pneumonectomy, as required, with the pleurectomy.

(Slide.) This is the specimen in a case in which an upper lobectomy and pleurectomy were performed, and shows the chronic empyema and the open bronchopleural fistula.

(Slide.) The next case is that of a young man with repeatedly negative sputum and gastric contents by all types of bacteriologic examination who, two years after development of a bronchopleural fistula following pneumonolysis, had a large residual tuberculous empyema with fever and malaise.

(Slide.) At exploration through the extrapleural mediastinal approach, the left lung was found to be uninvolved except for the upper lobe apical segment, which was shrunken and indurated. The uninvolved portions of the lung stripped with ease from the visceral wall of the empyema despite its two-year duration. Whether or not a lung is adherent to the visceral empyema wall is dependent on the presence and severity of disease in the lung. Uninvolved lung is usually not firmly adherent, and strips with ease. If the lung is healthy the visceral pleura is uninvolved, and I have been unable to find evidence of fibrosis and ingrowing trabeculae of fibrous tissue from the overlying empyema.

(Slide.) The apical segment was resected with the empyema, and when opened was found to contain a blocked cavity filled with caseous tuberculous material.

(Slide.) Several months later a small apical thoracoplasty closed the residual space, and almost the entire left lung was conserved and is healthy and functioning. This illustrates what may be accomplished by pleuropneumonectomy and its variations, in extirpating the pulmonary and pleural disease while permitting conservation of uninvolved lung in contrast to thoracoplasty and Schede resection of parietes which even at best cause unnecessary collapse and defunctionalization of uninvolved lung.

(Slide.) In some cases, as in this young girl, localized chronic empyemas, without significant associated active tuberculous or suppurative disease in the lung, may be resected without removing any lung. The intact excised empyema sac is shown. Such excision of chronic empyema removes the disease at once, conserves the lung function, precludes long periods of drainage and multiple operations, and obviates the need for thoracoplasty, since the underlying healthy lung can usually expand to fill the space.

(Slide.) This table presents part of my experience with pleuropulmonary resection for tuberculosis and empyema. This group of cases was presented last year at the National Trudeau Society, and I am presenting the same series now, omitting my more recent cases, so that each patient has been followed at least one and one-half years since operation. There were eighteen pleuropneumonectomies for destroyed lung with empyema, eight had obvious bronchopleural fistulas, and seven had thoracotomy or chest-wall sinus openings at the time of operation. Two patients had died when this series was presented last year and another has died since—the mortality, therefore, is 17 per cent, and 83 per cent are alive. One developed a chest-wall sinus, which has recently been excised. The others are well. Pleurectomy for empyema was associated with upper lobectomy in two cases, with upper lobectomy and lower lobe apical segmental resection in three cases, and with upper lobe apical segmental resection alone in one case. All six patients are alive and well. Of the total of twenty-four cases, three have died and twenty-one are alive; the recovery rate is 87.5 per cent, in contrast to the results of treatment by thoracoplasty and Schede. In addition, in one case, a chronic tuberculous empyema was excissed without any pulmonary resection and this patient is well.

DR. COTTON (closing).—I wish to reiterate that this paper is a preliminary report. In answer to Dr. Burnett's question, the survival rate for the malignant cases is four months to two years; the survival rate for the inflammatory cases is six months to four years. It was necessary to give up on many of the malignant cases. Fortunately, we have not had Dr. Burnett's experience in the one case of wound disruption. May I thank the discussants for their comments.

THE MANAGEMENT OF THE PATIENT WITH INOPERABLE CANCER OF THE CHEST

RICHARD H. MEADE, JR., M.D., AND RICHARD A. RASMUSSEN, M.D.
GRAND RAPIDS, MICH.

THE cure of the patient is obviously our chief aim in the treatment of any disease. This is no less true in cancer of the chest. However, it is common experience that the majority of patients with this disease are beyond the reach of our present methods of curative treatment when first seen by the surgeon. In 1948, Churchill,[1] in his excellent article on cancer of the lung, quoted the results reported by nine authors after 1940, in which 1,950 cases of cancer of the lung had been seen. In only 22 per cent were resections possible. More recent figures from various parts of the world show very little change (Table I), and our own experience in an area new to thoracic surgery has been similar. We have been able to do resections in 23.3 per cent of 116 patients. When we turn to cancer of the esophagus we find it impossible to discover the present operability rate. Much has been written in recent years about cancer of the esophagus, and operability figures have been given, but they are only in terms of the total number of operations and ignore the inoperable cases. The extremely low rate of operability found in 1942 by Santy, Ballivert, and Berard[2] in France, six of a total of seventy patients, is in striking contrast with the present report of Sweet's[3] experience with his private cases since 1944. He has seen 142 patients with cancer of the esophagus and has been able to do resections in ninety-two of them (65 per cent), but he adds that many of these resections were purely palliative. In our much more limited ex-

TABLE I. OPERABILITY OF CANCER OF THE LUNG

AUTHOR	TOTAL CASES	RESECTIONS	PER CENT
Mason* England (1949)	1,000	202	20.2
Gagnon† Toronto (1934-1945)	224	49	21.8
Overholt‡ (1932-1949)	721	200	27.7
Burnett§ (1936-1948)	527	94	17.8
Graham‖ (1945-1947)	211	58	27.0
Meade and Rasmussen (1947-1951)	116	27	23.3

*Mason, G. A.: Cancer of the Lung; review of 1,000 Cases, Lancet 2: 587-591, 1949.

†Gagnon, E. D.: Bronchogenic Carcinoma (12 Year Review and Operative Results), Canad. M. A. J. 58: 25-29, 1948.

‡Overholt, R. H., and Schmidt, I. C.: The Silent Phase of Cancer of the Lung. J. A. M. A. 141: 817-821, 1949.

§Burnett, W. E., Rosemond, G. P., Hall, J. H., and Caswell, T.: Carcinoma of the Lung, Arch. Surg. 61: 1052-1057, 1950.

‖Graham, E. A.: Considerations of Bronchiogenic Carcinoma, Ann. Surg. 132: 176-188, 1950.

Read before the Thirty-first Annual Meeting of the American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

perience, we have been able to do resections in nine of twenty-eight patients (32.1 per cent), seen in the last three years. We believe that this more clearly represents the true figure, as we are much more likely to see the hopeless cases than is Sweet. When one adds to these sites the mediastinum and chest wall, no improvement in operability is found. Thus, it is evident that in the majority of our cancer cases the problem is one of the management of the patient for whom no curative surgery is available. And again according to Churchill,[1] if we are to maintain our positions as doctors rather than as artisans, the responsibilities for the care of these patients must be faced. It is the purpose of this presentation to consider the basic principles involved, to cite three unusual cases of inoperable cancer in which unexpectedly good results were obtained, and to outline a plan for the management of these unfortunate patients.

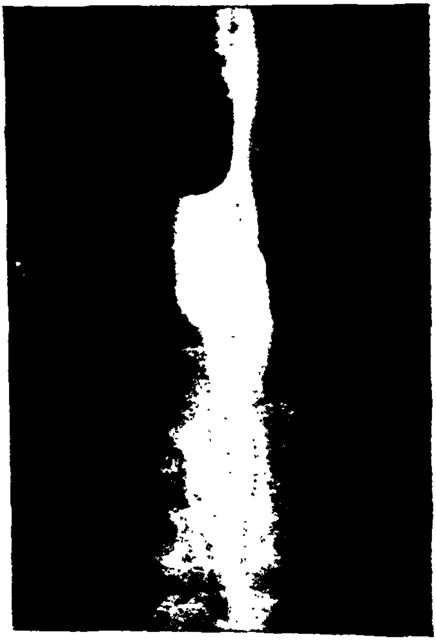

Fig. 1.—Esophagram of patient, Case 1, in February, 1948, showing narrowing of lower third of the esophagus.

There are many in this audience who can well remember when diabetes and pernicious anemia were in the same class with inoperable cancer today. The dramatic change in the outlook for diabetics following the discovery of insulin will never be forgotten by those who first had the opportunity of using this agent. Nor will it be hard to remember the introduction of liver therapy for pernicious anemia. Since then we have seen the great results obtained from the use of the sulfonamides, and the antibiotics, and the amazing changes produced by the use of cortisone and ACTH. How can one doubt the possibility of discovering some agent which will be effective in the treatment of cancer? It may not come in our lifetime, but we believe that it is of the utmost importance to impress upon the patient with inoperable cancer that such a discovery is a real possibility, and that it might come in time to help him. If we cannot cure our patients, we must not rob them of hope.

In the management of patients with cancer of the chest, the first step is of great importance. We must prepare the patient to accept whatever treatment is finally given as the best in his case. If he is told that he can be cured by the removal of his lung, and he later learns that his lung was not removed, he is left with no alternative but to believe that his case is hopeless. He must be told that while surgical removal is the quickest method of cure for the suitable case, there are certain types of cancer that can best be treated by other methods, and that the surgeon can only make the decision as to the best plan of treatment after exploring the cancer at the operation.

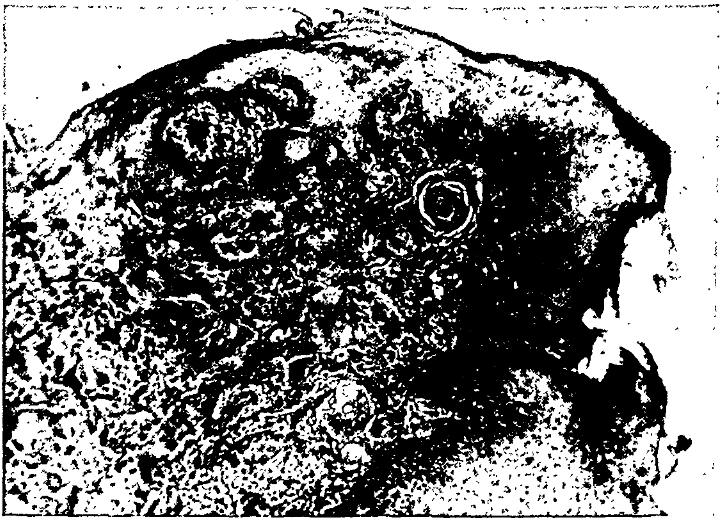

Fig. 2.—Biopsy of esophageal tumor in Case 1 showing a typical squamous-cell carcinoma.

While it is known that the duration of life with inoperable cancer is rarely as long as two years, everyone knows of a case in which all predictions for early death were proved false. There are occasional patients which, either without any specific treatment or after roentgen therapy, nitrogen mustard or other agents, live comfortably for years. It is helpful to tell patients with inoperable cancer about such cases, especially when they live in the same community, or are known to the patient. We have had a number of such experiences, and are briefly reporting here the stories of three of them.

The most striking example is the case of a 57-year-old man who was operated upon two years ago because of a carcinoma of the esophagus. The roentgenogram was typical (Fig. 1), and the biopsy (Fig. 2) showed a squamous-cell carcinoma. At operation the lesion was found to be resectable, but there were metastases in his liver and a hard mass in the pylorus. It was thought unwise to do a resection. A four-day course of nitrogen mustard* was given, and at six- to eight-week intervals his esophagus was dilated over a swallowed string by means of a Plummer bougie, making it possible for him to eat and to return to his former work. Once, ten weeks after operation, the string would not pass and it was necessary to pass it through an esophagoscope. His general condition has been excellent. Recent examination revealed a normal-looking man. The roentgenogram (Fig. 3) shows no evidence of the former esophageal lesion, and on esophagoscopy there was moderate narrowing but no tumor could be seen, and biopsies from the area showed no evidence of tumor.

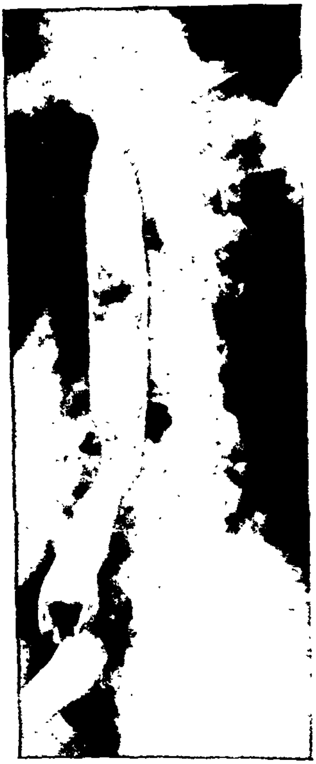

Fig. 3.—Esophagram of patient, Case 1, March, 1951, showing evidence of return to nearly normal appearance.

The second patient was a woman 82 years of age who, three years ago, was found to have a carcinoma of the upper esophagus. This involved the pyriform sinuses so that an esophagoscope could not be passed. A biopsy showed squamous-cell carcinoma (Fig. 4). The roentgenogram (Fig. 5) showed the obstruction and marked widening of the esophagotracheal space. A gastrostomy was done, and she was started on Teropterin and roentgen therapy.† The latter was soon discontinued because of edema. Two days later she was started on a four-day course of nitrogen mustard. Within two days she could swallow, and from then on her swallowing gradually returned to normal, although she had to chew her food well. Esophagoscopy three years later revealed marked narrowing so that the instrument could only be passed into

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*Nitrogen mustard dosage was 0.1 mg. per kilogram of body weight, given intravenously each day.

†Roentgen therapy for Case 2 amounted to 6,400 roentgen units through three portals.

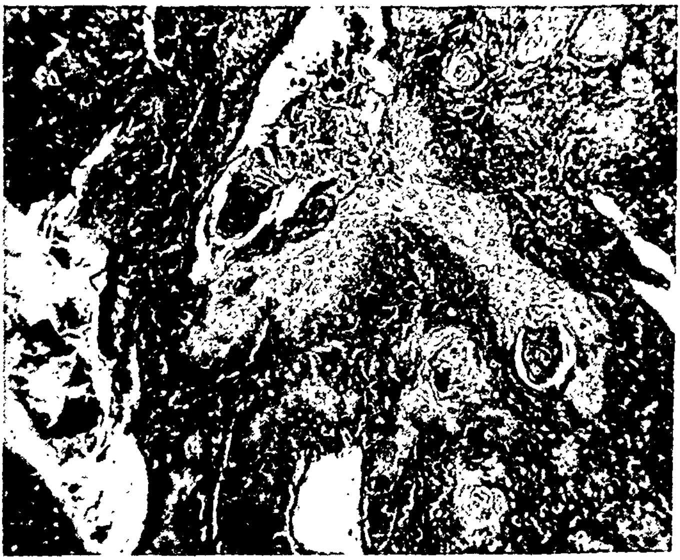

Fig. 4.—Biopsy of tumor filling upper esophagus of patient, Case 2, showing typical squamous-cell carcinoma.

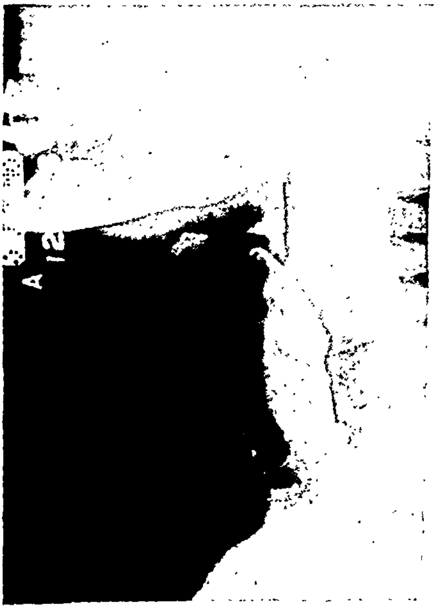

Fig. 5.—Roentgenogram in Case 2, showing marked deformity of upper esophagus and widening of the tracheoesophageal space.

the upper inch of esophagus, but the mucosa appeared normal. This was thought to be due to periesophageal fibrosis due to roentgen therapy. Fluoroscopic examination revealed no abnormality, but the barium passed too rapidly to allow good films to be taken.

The third patient was a 60-year-old man who had a cough and some dyspnea. Roentgenograms (Figs. 6, 7, and 8) showed evidence of marked pressure on the anterior tracheal wall, and bronchoscopy revealed the trachea to be flattened to a narrow transverse slit with roughening and induration of the mucosa and a broad carina. No biopsy was taken for fear of producing tracheal obstruction. He was told that he had cancer which could not be treated surgically, but that he would be given nitrogen mustard. As he knew the patient with cancer of the esophagus who had apparently been so dramatically helped by nitrogen mustard, he was strongly reminded of her case. He received a four-day course of nitrogen mustard without noticeable effect. Later he was given a second course of nitrogen mustard in preparation for roentgen therapy, as it had been pointed out by Karnofsky[4] of the Memorial Hospital, New York, that this would prevent the development of edema. Roentgen therapy was started two days later and continued without incident until he had received 10,000 roentgen units. He improved greatly and carried on his work. Later an x-ray film showed a nearly normal trachea (Fig. 9) and a negative routine chest film. One month later he developed slight ankle edema and

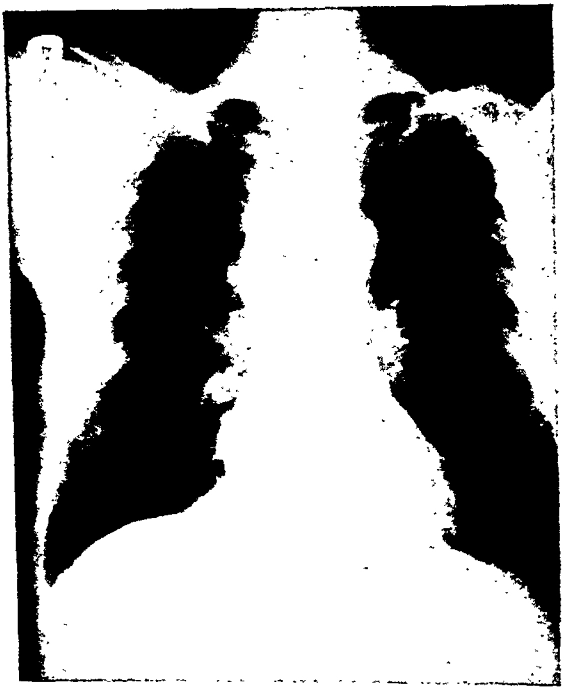

Fig. 6.—Posteroanterior roentgenogram of the chest of patient, Case 3, showing no obvious abnormality.

recurrence of dyspnea. On fluoroscopy the heart was markedly enlarged, and he was admitted to the hospital, where he died ten days later. Autopsy showed extension of his carcinoma downward into the mediastinum and into the heart. The trachea, however, was not narrowed.

It is not the object of this article to discuss the value of the forms of treatment used in the three cases just presented, as it is realized that many others have had similar experiences. However, we believe that certain features are worth emphasizing. The first is the use of nitrogen mustard immediately to precede roentgen therapy in patients who might suffer from the development of edema. This effect was dramatically illustrated in our third case where edema might have proved fatal. The other form of therapy which we believe worth stressing is the use of dilatation of the esophagus in patients with inoperable cancer. The passage of a Plummer bougie over a previously swallowed string rarely causes any trouble, and makes it possible for many of these patients to continue to swallow food up to the time of their death. Vinson,[5] and other bronchoesophagologists, have stressed the great value of this procedure for many years, but it is rarely even mentioned in articles by others. In our limited experience we have been greatly impressed with its value.

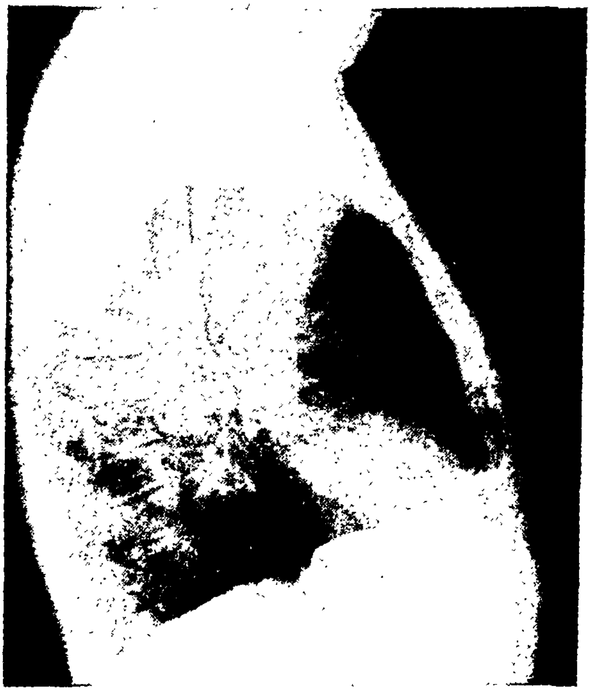

Fig. 7.—Lateral roentgenogram of patient, Case 3, showing evidence of tumor causing marked pressure on anterior surface of the trachea.

All of these patients were told that they had carcinoma. They were told that their carcinomas could not be treated surgically but that other forms of

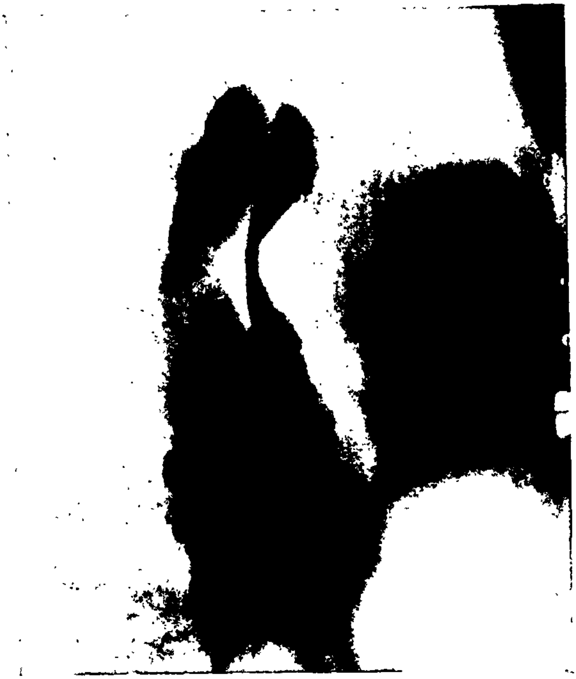

Fig. 8.—Lateral tomogram of patient, Case 3, showing evidence of tumor, causing marked pressure on the anterior surface of the trachea.

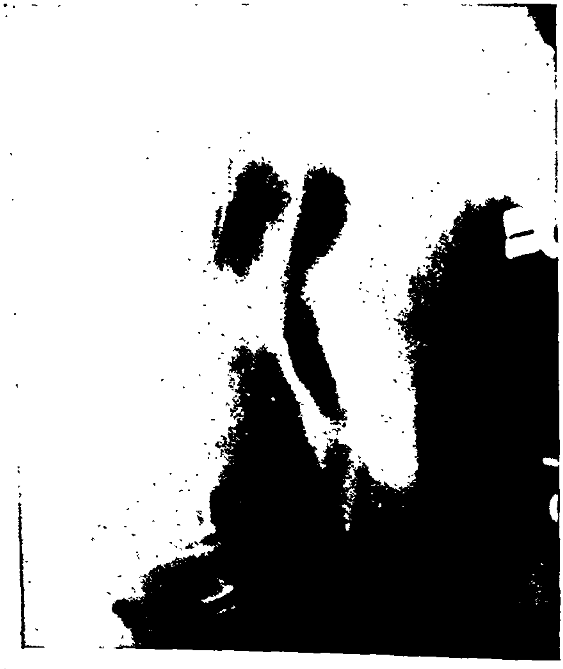

Fig. 9.—Lateral tomogram of patient, Case 3, following nitrogen mustard and roentgen therapy, showing nearly normal tracheal lumen.

treatment would help them, and that they had to hope that they might be cured. The one patient who has died was able to lead a normal life up to ten days before his death, even though he lived only four months after the tumor was first recognized. Two of these three patients are still alive and have no evidence at present of their former carcinoma.

The problem of how much to tell a patient with inoperable cancer is not a simple one, and we do not believe that any rigid rules can be made. There is evidence that cancer patients in general do want to know the truth about themselves. The studies[6] made at the University of Minnesota showed that practically all of the cancer patients questioned there were emphatic in their desire to know the truth about their own conditions, and that they believed the great majority of all cancer patients should be told the truth. However, this study was concerned with the reaction of the patient to the knowledge that he had cancer, not that he had an inoperable tumor. Certainly, the information about inoperability should not be given until the patient has recovered from his operation. To give it earlier might prove to be an unbearable shock. The intelligence of the patient, his financial position and the character of his family are all important factors. To withhold this vital information from the head of a family might prove disastrous to the family. However, we are discussing the subject only from the standpoint of the patient. Will he be happier knowing the full truth? If he has no symptoms, or can explain those he has on the basis of some obvious cause, he will live happily without the knowledge of his condition. If he has symptoms that cannot properly be explained without telling him the truth, he will worry more and more about himself, and become convinced that his doctor does not realize how sick he is. He will lose confidence in his doctor and will find fault with everyone dealing with him. Those who are told the truth, in our experience, have at once changed their attitudes and have spent their last days in peace.

Although the patients upon whom most of us operate are referred by other physicians, and after operation are referred back to them, the responsibility for their fundamental care should remain with the surgeon. He it is who makes the decision as to the definitive treatment. We believe he, rather than the family doctor, should tell the patient of his condition. It is frequently impossible for the surgeon to follow his patients in person, but we believe that his responsibility as a consultant does not end with the patient's discharge from the hospital. The patient with inoperable cancer who is discharged by his surgeon must feel that his death sentence has been sealed. The knowledge that his surgeon is still interested in his case, and is on the alert to bring him any hope, will give him a sense of security and an optimistic outlook that will be of great value.

CONCLUSIONS

Because the great majority of patients with cancer of the chest are not seen early enough to allow for curative surgery, their management is obviously one of the greatest responsibilities of the thoracic surgeon. As the use of various forms of nonsurgical treatment have at times proved of value, we believe they should be used in most cases of inoperable cancer. Furthermore, we believe that in general all patients with inoperable cancer of the chest should be told the basic truth, but that hope for their recovery must never be destroyed.

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DISCUSSION

DR. RICHARD H. MEADE, Jr., Grand Rapids, Mich.—It may seem strange to you that at this meeting where we are discussing scientific subjects and discussing definitive treatment of patients, we should introduce the subject of the management of the patient with inoperable cancer. So far as I can remember, the last time any speaker before this organization mentioned the patient as an individual was in 1934, when Rudolph Jessen of Switzerland discussed the treatment of bilateral tuberculosis by means of thoracoplasty. Unfortunately, the discussion of that paper was not published, but those of us who heard it will never forget the beautiful tribute that Dr. Lambert paid to Dr. Jessen. I am sure that all of us are impressed with the large number of patients with cancer for whom we have no definitive treatment. However, we must remember that they are individuals, and that we owe something to them even though we cannot cure them of their disease.

DR. DEAN B. COLE, Richmond, Va.—I am glad to hear this excellent paper, but do not agree that all patients with cancer wish to know or should know the diagnosis. We see a number of patients who probably suspect they have cancer, but never ask.

I think it a mistake for the surgeon to be in too great a hurry to inform the patient of his diagnosis, and I think he should be especially careful if the referring doctor is to take care of the patient during the remainder of his illness. It is easy to be scientifically cold-blooded in passing a death sentence, even though some hope may be held out for a reprieve. I think the family should always know the diagnosis. Every possible consideration should be given the patient in talking to him or his family.

DR. GABRIEL P. SELEY, New York.—I would like to report an experience which we have had with three cases recently, which deals with this subject. The first was a 62-year-old man who was operated upon on the private service of Dr. Aufses. This man had a positive biopsy from the trachea and right main bronchus for squamous-cell carcinoma. At the insistence of the medical man this patient was treated with radiotherapy by the grid technique. Following a course of radiotherapy, bronchoscopy showed the trachea and bronchus both negative. Operation was performed, a total pneumonectomy was done, and at that time a tumor was noted in the lung, but the mediastinum was clear. The pathologic specimen showed a squamous-cell carcinoma of the lung with no involved lymph nodes, and there was still residual tumor in the upper lobe bronchus. This man developed a rather indolent type of postoperative wound infection and empyema, but has done fairly well.

The second patient was a man 65 years of age, who was operated upon on the service of Dr. Touroff. This man had a positive biopsy for carcinoma of the left upper lobe bronchus, small-cell in this case. He had a large mass in the lung and was treated by radiotherapy in the same manner. The lung mass decreased in size. Bronchoscopy showed a negative bronchial biopsy. He was operated upon; a pneumonectomy was performed. At that time it was noted that he had a mass in the lung but the mediastinum was completely free. In this case numerous and careful examinations of the specimen removed have failed to show any evidence of carcinoma. Encouraged by these two results, a third patient was treated in a similar manner. He was a 61-year-old man who had a six-month history of carcinoma of the lung. He had a positive biopsy, both from the carina and the right upper lobe bronchus, for squamous-cell carcinoma. He was given radiotherapy. He was then re-bronchoscoped, and the carina was negative. A pneumonectomy was performed, a right pneumonectomy, at which time a mass in the lung was noted, but the hilus was free. The pathologic specimen showed squamous-cell carcinoma of the lung, but no evidence of endobronchial lesion and no involved lymph nodes.

Both the latter two patients had primary union of their wounds, and both have done well. This, I think, is another method which may be considered, and may be employed in order to make some of these patients who seem to be inoperable, operable.

DR. ROBERT R. SHAW, Dallas, Texas.—In discussing this always timely paper by Dr. Meade and Dr. Rasmussen, I would like to add two instances to the rays of light in the dark corner.

(Slide.) This is a patient whom I saw in October, 1945. I am indebted to the Scott and White Clinic of Temple, Texas, for the first film. This large shadow in what proved to be the anterior portion of the left chest was thought to be a mediastinal dermoid tumor, but on exploration I found that it was a large mass arising in the left upper lobe which had invaded the pericardium. I opened the pericardium and found approximately 300 c.c. of bloody fluid in the pericardial sac. It was more expedient to take the biopsy from within the pericardial sac than at any other part of the tumor mass, so biopsy was taken from that point. The chest was closed. The situation was explained to her husband, that it was inoperable, and that the prognosis was very bad. I did advise, however, that she have deep x-ray therapy. This was given, and in the second film you see the result a little more than five years later. This patient has had absolutely no recurrence of symptoms, and is leading an entirely normal life. The only complaint is that she is a bit overweight.

(Slide.) This is the film of a 54-year-old man whom I saw in December, 1945, now in his sixth year. The bronchoscopy was positive for epidermoid carcinoma, the biopsy being taken from the right upper lobe orifice. I advised exploration, but at exploration I found that the carcinoma, although not large, had extended up the tracheal wall so that I thought it impossible to remove it safely. The phrenic nerve was involved in the tumor, and I cut that because he was having intractable shoulder pain. After exploration, the usual bad prognosis was given to the wife, but I did advise that he have x-ray therapy.

In the next film you see the result two months following a course of x-ray therapy that took approximately three weeks to give.

(Slide.) Here we see the result six months later when the right upper lobe had completely broken down in suppuration. He was a thoroughly miserable man, coughing out purulent sputum that was foul, and running temperature elevation. But gradually he began to improve, and the last film shows the appearance of his chest for the past three years. It has not changed in three years' time. At the time exploration was done he had just been re-elected mayor of a small city in east Texas. I made a mental note that he would not live out his two-year term. He did live out his two-year term, but he was not feeling too well at that time so he did not run for re-election. However, at the last election he did run, and is now mayor of the city and is feeling very well. I am getting quite weary of telling his wife that this man can't live very long.

Of course these are exceptions to what we usually see, but I happen to live in a community where the radiologic pace is set by a man who believes in carrying x-ray therapy to safe limits. In other words, he gives them a good big dose, and the other men working in the community do the same. It is rather strange to have heard the two very fine presentations this morning telling of the extension of the surgical attack upon this disease, and then think of the plea made by the usual surgeon to the radiologist, not to make these patients too sick with their x-ray therapy, "and please don't burn the skin, because we just want to palliate the cough a little bit." Sometimes I believe if x-ray therapy is carried to its logical conclusion, an amazing result may be obtained.

DR. PAUL HOLINGER, Chicago.—This excellent paper by Dr. Meade and Dr. Rasmussen certainly raises some fundamental points and indicates, in a way, a justification for a subspecialty of broncho-esophagology. The surgeon doing endoscopic work is primarily interested in the diagnostic phases of the field: the endoscopic management of the patient requiring a nonsurgical course of treatment frequently reverts to the otolaryngologist who is doing endoscopic procedures, to the broncho-esophagologist. This was emphasized in Dr. Olsen's discussion, when Dr. Tuttle mentioned that surgery may be necessary in benign stenotic esophageal lesions because one cannot get anybody to dilate them. Dr. Olsen, you recall, answered that very well by simply stating that for a man who could take out the auricles of the heart, dilatation of the esophagus should not be too difficult.

Dr. Meade has stressed the fact that patients with inoperable esophageal carcinoma can be treated palliatively by dilatation and by roentgen therapy. We have used a resectoscope technique using a cystoscopic type of resectoscope to resect the carcinomatous tissue when the lesion is inoperable, thus permitting the patient to swallow in a normal manner. In inoperable carcinoma of the tracheobronchial tree the removal of tumor tissue from the trachea with the resectoscope is feasible. Radon seed implantation of tumors likewise should not be overlooked in otherwise inoperable patients. Two- to three-year alleviation of symptoms in patients with inoperable esophageal cancer is not at all uncommon under such management, and in one case such treatment permitted normal swallowing for six years. In the tracheobronchial tree, such management of tracheal tumors is particularly noteworthy. Repeated endoscopic resection of carcinomatous tissue in one inoperable cancer of the trachea was done for seven years, stressing the value of endoscopic management of the patient with inoperable cancer.

The danger of such reports is that the broncho-esophagologist is accused of keeping patients from the surgeon when the lesions are resectible. This is not the case. The broncho-esophagologist is just as desirous to refer the patient for surgical removal of these lesions when such removal is possible, as the surgeon is to see the patient. I think, however, that the management of the inoperable patient which Dr. Meade and Dr. Rasmussen have stressed, should continue with the endoscopic procedures that have been mentioned.

MR. W. P. CLELAND, London, England.—I have been most interested in this discussion, and have been prompted to tell you a little of my own experiences in the field of carcinoma of the bronchus in connection with x-ray therapy. Three years ago a man, aged 40 years, with carcinoma of the bronchus, came to me, and when I bronchoscoped him I found a tumor arising in the right upper lobe, extending up the right main bronchus practically to the carinal level. There was marked subcarinal widening and I considered that, bronchoscopically, he was inoperable. A biopsy showed an oat-cell carcinoma, and I referred him to an x-ray therapist who gave him a radical course of x-ray therapy spread over a period of about six weeks. I saw him again about four months later, and his improvement was dramatic. The solid upper lobe was completely aerated, he had put on 30 pounds in weight, and when I bronchoscoped him a second time there was no evidence of tumor in the bronchus. I subsequently performed a radical pneumonectomy without any difficulty. The specimen failed to reveal any macroscopic or microscopic evidence of tumor, but two years later the man died from secondary deposits.

This prompted me, about eighteen months ago, at the Postgraduate Medical School in London, to start a clinical experiment combining preoperative x-ray therapy with resection. During the first twelve months of that experiment 175 patients were referred for assessment, and fifty of those were considered to have only a primary lesion without obvious secondary deposits either clinically or with x-rays. The interesting thing about these cases which were all treated radically with x-ray therapy has been the tremendous improvement shown by the majority of these people. The tumor masses in many cases have disappeared, the lung has re-aerated, and improvement in the general condition has been most striking. Twenty-four pneumonectomies have been performed to date on these cases, and the operative difficulties were not any greater than in the untreated cases.

I would like now to mention a small group of twelve cases who were regarded, before x-ray therapy, as being bronchoscopically inoperable. That means either that the growth was too close to the trachea, or there was widening below the carina, to justify a pneumonectomy. Five of those patients subsequently became resectable, and pneumonectomies were performed. Their subsequent course has not been very different from the normal pneumonectomy, although one patient has since died from a recurrence in the stump. It is far too soon to decide whether this combined treatment is going to be an advance with a longer survival rate, but I think the experiment is worth continuing.

I do not think any mention has been made this morning of the palliation of carcinoma of the bronchus, or treating superior mediastinal obstruction or secondary deposits in bone; both of the latter respond extraordinarily well to x-ray-therapy, with considerable symptomatic improvement, even though it is temporary.

DR. RASMUSSEN (closing).—We wish to thank the discussers. With regard to Dr. Cole's remark, we did not mean to imply that he too should not talk to his patient about this terrible condition, because he undoubtedly will have more contact with him, but it is sometimes helpful if both physician and surgeon talk to the patient.

It is probably unwise to make predictions as to the time of death lest the radiologist or someone else afford the patient the advantage of an unusual outcome.

RADIOACTIVE ISOTOPES IN THE PALLIATIVE MANAGEMENT OF CARCINOMATOSIS OF THE PLEURA

Edward M. Kent, M.D., and Campbell Moses, M.D. (by invitation)
Pittsburgh, Pa.

CARCINOMATOSIS of the pleura associated with a profuse effusion represents one of the complications of malignant neoplasms which is encountered with some frequency. The clinical management of such complications has always been difficult and unrewarding if the effusion formed rapidly. Victims of this unfortunate situation suffer from recurring, severe dyspnea as the pleural space inexorably fills with fluid after each aspiration. The loss of large quantities of protein into the effusion increases the wasting and hastens death materially. Few individuals live more than weeks once carcinomatosis of the pleura becomes established, provided an intractable, rapidly reforming effusion is also present.

The treatment of such effusions has been ineffective in the past. In our experience efforts to stop or delay the formation of pleural fluid have been to little avail. Roentgen therapy has offered the most benefit, but failure has been experienced in the great majority of patients given irradiation therapy by this means. Some reasons for this failure are obvious. One cannot irradiate adequately the entire hemithorax of an individual, nor can the rays be directed accurately at the areas of pleural implantation, since their precise location is unknown and they are usually numerous and scattered widely on both surfaces of the pleural cavity.

Radioactive isotopes were considered as a possible source of irradiation therapy under such circumstances, inasmuch as these substances can be injected directly into the pleural effusion and thereby irradiate all surfaces of the cavity. Because of the relatively short range of beta rays in tissue (0.8 to 2 mm. for the beta rays from I^{131}), it was thought that relatively large doses of radiation could be delivered to the pleural surface with a minimum of radiation damage to adjacent structures.

At the time this study was begun, no data were available on the absorption, excretion, stability, or toxicity of radioactive material administered by the intrapleural route. However, previous experience by several groups of workers using large doses of I^{131} in the treatment of thyroid carcinoma had established the safety of this procedure both to the patient and attending personnel. For these reasons, and because of its ready availability, carrier free I^{131} in weak basic sodium sulfite solution as supplied by the Oak Ridge National Laboratory was initially used in this study. As this work progressed, radioactive colloidal

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Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

gold (Au^{198}) and I^{131} tagged human albumin became available* for investigational use and these isotopes were used in a limited number of patients in this series.

MATERIAL AND METHODS

Only patients with neoplastic disease and established carcinomatosis of the pleura who demonstrated a rapidly forming, intractable pleural effusion were employed for these preliminary observations. In most instances (14) the neoplasm was of bronchogenic origin, in some (4) the primary tumor had been carcinoma of the breast, and in one patient the primary neoplasm was a fibrosarcoma of the abdominal wall which had grown to enormous size over a period of seventeen years and had resulted in extensive intrathoracic metastases with intractable pleural effusion.

An arbitrary dosage of 10 millicuries of I^{131} was chosen initially for this investigation, but this dosage was later increased to as much as 30 millicuries. The I^{131} as obtained from Oak Ridge was transferred to a hot, sterile screw cap bottle of 50 to 100 ml. capacity. This preparation was found to be bacteriologically sterile. Under local anesthesia the pleural effusion was aspirated as completely as possible. During the aspiration the I^{131} containing bottle was filled with pleural fluid. At the conclusion of the aspiration the I^{131} containing pleural fluid was reinjected into the pleural cavity. This fluid was aspirated and reinjected several times in an attempt to deliver all of the isotope into the pleural space.

Immediately after completing the injection the operator thoroughly rinsed the gloves, syringe, needle, tubing, and stopcock in cold running water. If this washing is promptly and thoroughly done no significant contamination can be detected on the equipment.

In most of the patients here reported the urinary excretion of isotope in the two to five days following its administration was determined. Details of this excretion study will be provided in a separate report. However, of the twelve patients receiving I^{131} in this series on whom excretion data was obtained, 62 per cent of the administered I^{131} was retained forty-eight hours. Although accurate quantitative determinations of thyroid uptake were not made, undoubtedly much of this activity was retained in this gland after removal from the pleural space. In Case 10, five days after the administration of 25 mc. of I^{131}, only eight per cent of the administered radioactivity remained in the pleural fluid. When this same subject was given 5 mc. of I^{131} tagged albumin 93 per cent was retained in the body five days after administration and 72 per cent was retained in the pleural fluid. A similar study with radioactive colloidal gold in Case 11 revealed only 0.47 per cent of the gold appearing in the urine in seventy-two hours, and 23 per cent was retained in the pleural fluid on the fifth day.

RESULTS AND CASE REPORTS

The results of therapy, the isotopes with the doses employed, and the types of carcinoma present are indicated in Tables I and II. Analysis of these tables

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*Through the courtesy of D. L. Tabern, Ph.D., Abbott Laboratories, Chicago, Ill.

TABLE I. SUMMARY OF DATA AVAILABLE IN THE STUDY OF FOURTEEN PATIENTS WHOSE PRIMARY CARCINOMA AROSE IN THE LUNG

CASE NO.	TYPE OF CARCINOMA	ISOTOPE AND DOSAGE	RESULTS
1	Undifferentiated	10 mc. I^{131}	Fluid checked but observation limited to 21 days by death
2	Epidermoid	10 mc. I^{131}	Materially improved. Aspiration only once in two months of life following treatment
3	Squamous-cell	10 mc. I^{131}	Fluid completely checked. Now alive more than 12 months
4	Undifferentiated	20 mc. I^{131}	Effusion materially retarded—Aspirated twice in 3½ months of life after therapy—total 3100 c.c. Previously aspirated twice weekly average of 2700 c.c. each time
5	Undifferentiated	10 mc. I^{131}	Effusion completely checked. Lived 8 months
6	Squamous-cell	10 mc. I^{131}	Effusion completely checked. Lived 10 months
7	Undifferentiated	30 mc. I^{131}	Died 17 days after therapy. Cannot be evaluated
8	Squamous-cell	37.6 mc. Au^{198}	Effusion checked. Only 6 weeks follow-up
9	Adenocarcinoma	19.2 mc. I^{131}	Died 15 days after therapy. Cannot be evaluated
10	Adenocarcinoma	5 mc. albumin I^{131} 55 mc. I^{131} 120 mc. Au^{198}	Most refractory to treatment of all cases. Effusion very materially slowed. Now 4 months post treatment
11	Undifferentiated	40 mc. Au^{198}	Died 13 days after therapy. Cannot be evaluated
12	Squamous-cell	2.475 mc. I^{131} in serum albumin	Effusion completely checked. Alive 6½ months
13	Squamous-cell	20 mc. I^{131}	Effusion completely checked. Died 2 months
14	Undifferentiated	20 mc. I^{131}	Effusion completely checked. Alive 4 months

TABLE II. SUMMARY OF DATA IN THE STUDY OF FOUR PATIENTS IN WHOM THE PRIMARY CARCINOMA AROSE IN A BREAST AND OF ONE PATIENT WHOSE PRIMARY TUMOR WAS A MYXOFIBROSARCOMA OF THE ANTERIOR ABDOMINAL WALL

CASE NO.	TYPE OF CARCINOMA BREAST	ISOTOPE AND DOSAGE	RESULTS
15	Adenocarcinoma	10 mc. I^{131}	Completely checked effusion. Died in 4 months
16	Adenocarcinoma	20 mc. I^{131} each pleural cavity	Bilateral pleural effusion. No further aspirations required after therapy but pleural spaces were not dry. Died 3½ months after therapy
17	Adenocarcinoma	30 mc. I^{131}	Completely checked effusion. Alive 2 months
18	Adenocarcinoma	18.58 mc. I^{131}	No further aspirations required. Pleural cavity not dry. Died 4 months after therapy
19	Myxo-fibrosarcoma	10 mc. I^{131}	Completely checked effusion. See case report. Died 6½ months after therapy

shows that of the fourteen patients shown in Table I, three died too soon after therapy to be useful in evaluating results. However, in no instance was the pleural effusion a factor in the death of the patient, and the effusion in each case appeared to be checked or greatly retarded. In three instances, the results would be classified as fair since fluid did reaccumulate in sufficient quantity to require aspiration at least once after the immediate post-therapy interval. In eight patients, the effusion was promptly and completely checked. The results shown in Table II are those obtained in four patients, all women, who had the primary carcinoma in a breast, and in one patient whose primary lesion was a myxofibrosarcoma of the anterior abdominal wall. The results were good in each instance, although two of the patients continued to have small amounts of pleural fluid until death.

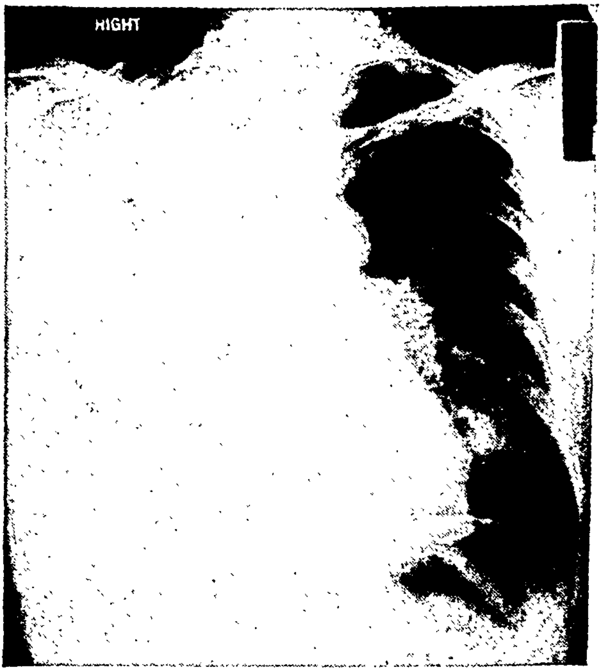


Fig. 1.—Chest film demonstrating right pleural effusion in patient designated in text as Case 1.

A brief description of the first patient treated by this technique in this investigation is presented: *J. W., a white man 62 years of age*, was admitted to the hospital complaining of severe dyspnea. The clinical findings were those of right-sided pleural effusion, and these were confirmed by a roentgenogram of the chest (Fig. 1). Thoracentesis yielded 500 c.c. of bloody pleural fluid, and at the *conclusion of the aspiration the pleural space had not* been emptied. Reaccumulation of the pleural fluid was so rapid that aspiration was required at frequent intervals and 4,230 c.c. were removed over a period of seven days. Carcinoma cells were found to be present in the pleural fluid (Fig. 2), and upon bronchoscopy a neoplasm was seen in the right *intermediate* bronchus (Fig. 3). Biopsy of this lesion revealed an undifferentiated carcinoma. On the fourteenth hospital day a single dose of 10 mc. of radioactive iodine was injected into the right pleural cavity, and studies of the rate of excretion of the isotope revealed that 64 per cent had been excreted in the urine during the first forty-eight hours immediately following injection. While our first reaction to this observation was one of disappointment and belief that the isotope had been excreted too quickly to have been effective, such was not the case. The patient did not again present signs and symptoms indicating a need for thoracentesis. However, numerous subsequent aspirations were performed for study purposes. During the first five days after treatment 4,450 c.c. of blood-tinged fluid were removed from the pleural space, and on the succeeding days until death "dry taps" were experienced. Twenty-one days after treatment the patient died, and an autopsy was performed. Although there were extensive metastases to the surfaces of the pleural cavity, only a small volume of fluid was present. Microscopic examination of the metastatic implants on the pleura revealed cytological changes in the superficial layers of carcinoma cells indicative of cell death (Fig. 4). Additional autopsy findings included the presence of extensive metas-

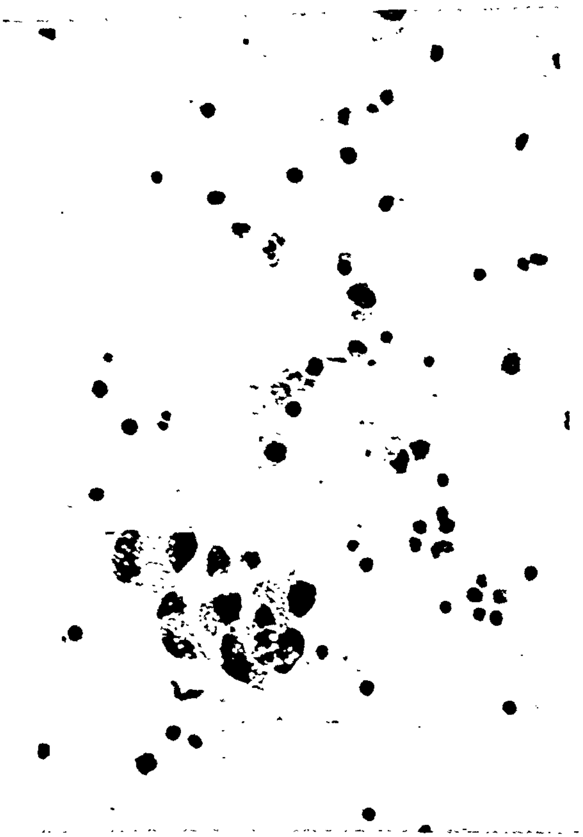


Fig. 2.—Carcinoma cells in the pleural fluid. Case 1. (×510.)

tases to the peribronchial and mediastinal lymph nodes, the periaortic lymph nodes, the diaphragm, the liver, the right adrenal, and to the celiac lymph nodes.

A typical good result can be shown by the presentation of Case 2: A 58-year-old woman who had been the victim of an intractable right pleural effusion was admitted to the hospital with a history of repeated thoracentesis during the five weeks immediately preceding hospitalization. A total of 18,000 c.c. of blood-tinged pleural fluid had been removed, and cytological studies of the fluid revealed carcinoma cells (Fig. 5). Upon admission to the hospital the right pleural space was filled with fluid (Fig. 6), and the patient was suffering pronounced dyspnea and displaying moderate cyanosis. Bronchoscopy revealed a neoplasm which partially obstructed the right lower lobe bronchus, and a biopsy revealed carcinoma (Fig. 7). A single injection of 10 mc. of I^{131} was followed by a cessation of the effusion, and further aspirations were not required. The patient lived for nine and one-half months, and autopsy confirmed the diagnosis of bronchogenic carcinoma with metastases to the pleura.

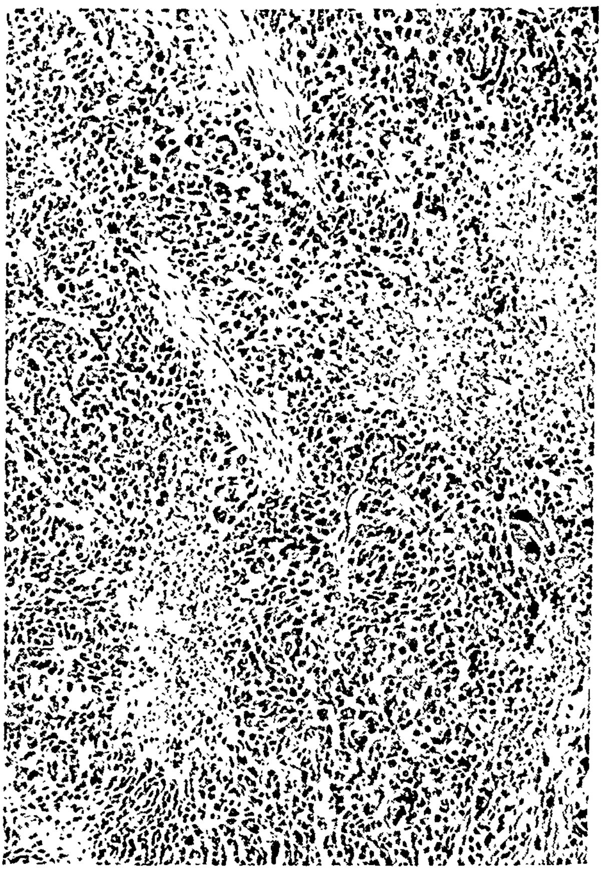
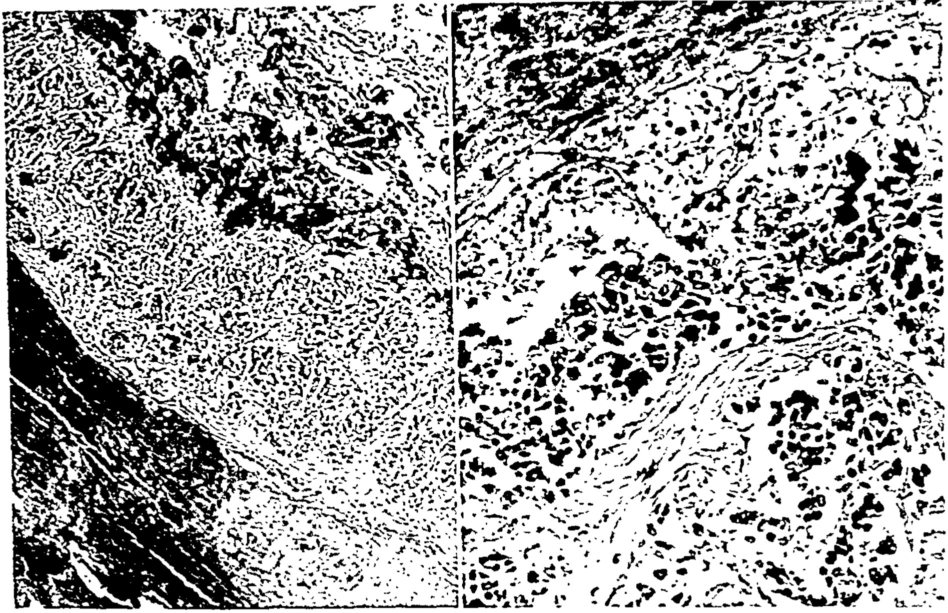


Fig. 3.—Bronchoscopic biopsy from patient in Case 1 revealing undifferentiated carcinoma. (×150.)



A. *B.*

Fig. 4.—*A.* Photomicrograph of pleural surface specimen removed at autopsy in Case 1. Changes in the surface cells of the carcinomatous implants are visible. (×75.)

B. Higher power photomicrograph of surface cells of carcinomatous implant. (Autopsy specimen, Case 1, ×275.)

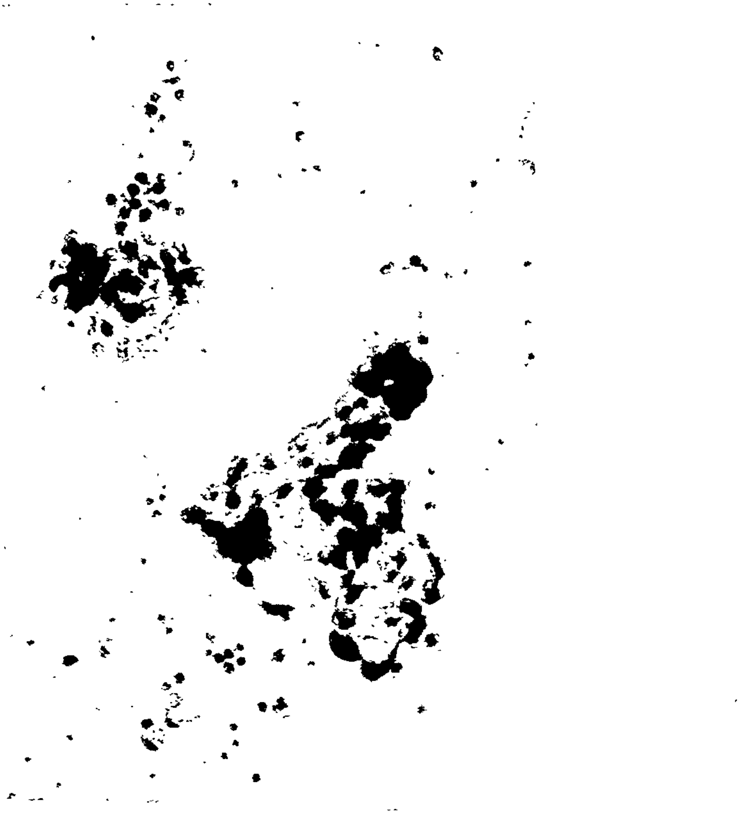


Fig. 5.—Carcinoma cells found in pleural fluid, Case 2. (×540.)

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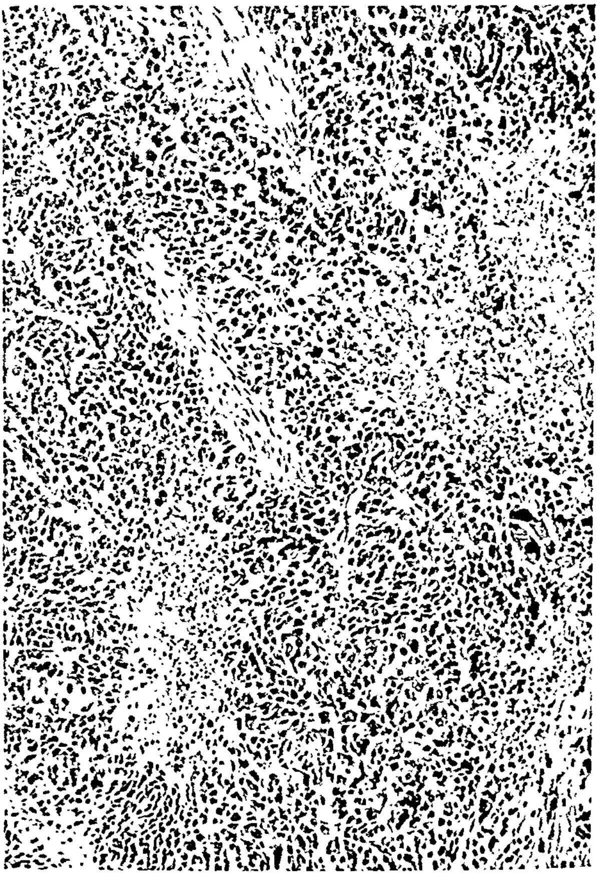
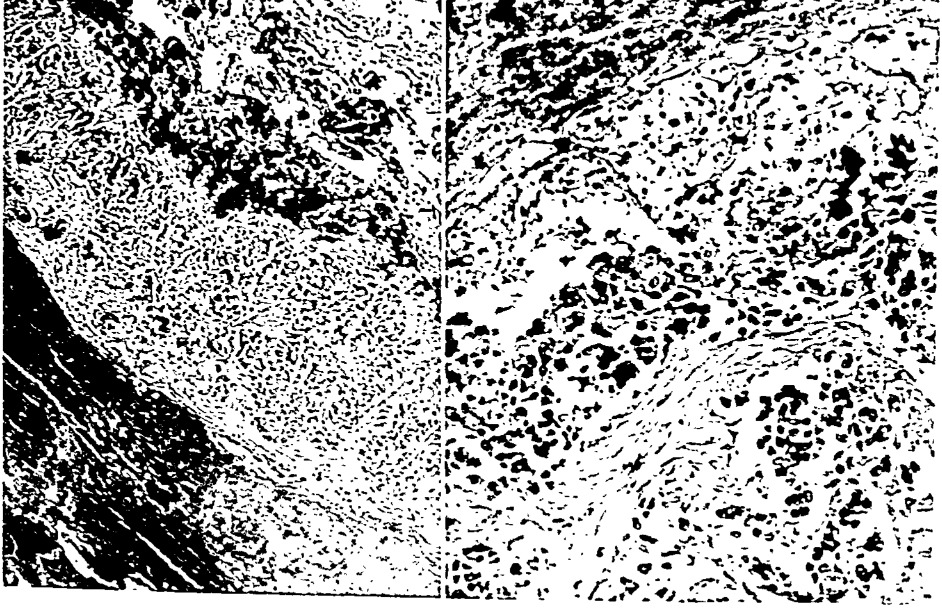


Fig. 3.—Bronchoscopic biopsy from patient in Case 1 revealing undifferentiated carcinoma. (×150.)



A.

B.

Fig. 4.—*A*. Photomicrograph of pleural surface specimen removed at autopsy in Case 1. Changes in the surface cells of the carcinomatous implants are visible. (×75.) *B*. Higher power photomicrograph of surface cells of carcinomatous implant. (Autopsy specimen, Case 1, ×275.)

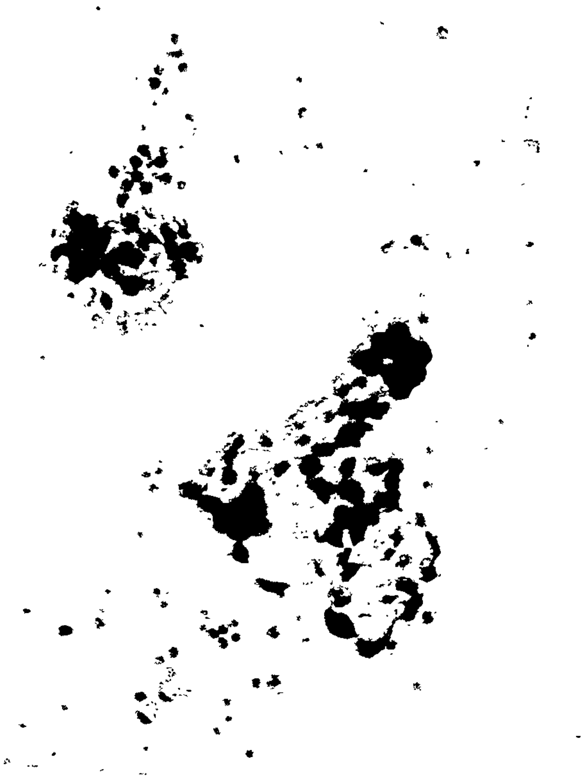


Fig. 5.—Carcinoma cells found in pleural fluid, Case 2. (×540.)

Case 3 is presented because of the unusual history and because of the fact that this patient presented a very large myxofibrosarcoma of the abdominal wall which had metastasized to the lungs and pleura. The patient, a 66-year-old man, was admitted to the hospital complaining of severe dyspnea and orthopnea. Physical signs and a chest film (Fig. 8) indicated the presence of a massive left pleural effusion and a total of 7,000 c.c. was aspirated during the first four days of hospitalization. A very large, nodular, firm mass was present on the anterior abdominal wall which was not fixed to the muscles, and which appeared to have arisen in the subcutaneous tissues. The patient first noticed a small mass at this point seventeen years previously, and gave the history that it had grown slowly during the interval to reach great size, but there never had been pain or other discomfort. This mass was removed and was found to be a myxofibrosarcoma of the subcutaneous layer (Fig. 9). In an effort to check the rapidly forming pleural effusion, 10 mc. of I^{131} was injected into the left pleural space. No further aspirations were necessary and the pleural fluid disappeared, revealing a large intrathoracic mass on the roentgenogram (Fig. 10). The patient resumed his occupation and worked for six months. At the end of this period, a rapid decline took place, and death occurred in two weeks. Autopsy findings demonstrated the presence of the massive tumor in the left lung (Fig. 11), multiple smaller metastases in both lungs, and widely disseminated metastases throughout the body. Microscopic examination of these masses (Fig. 12) led to the opinion that they represented metastases from the abdominal

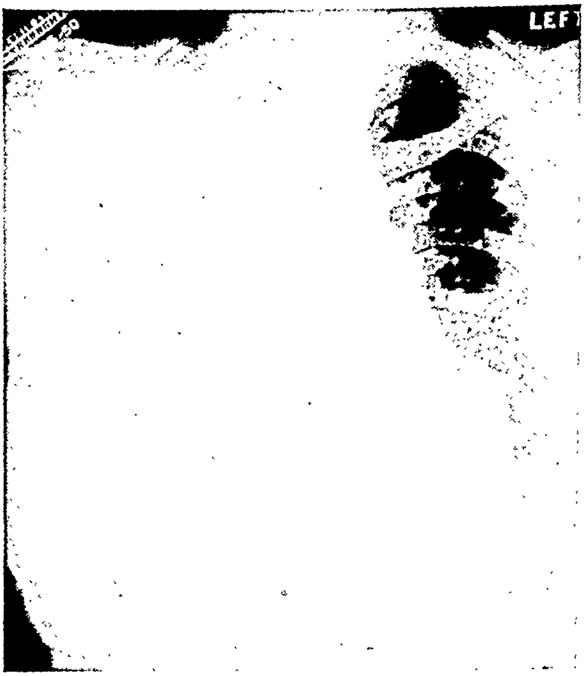


Fig. 6.—Chest film demonstrating massive right pleural effusion, Case 2.

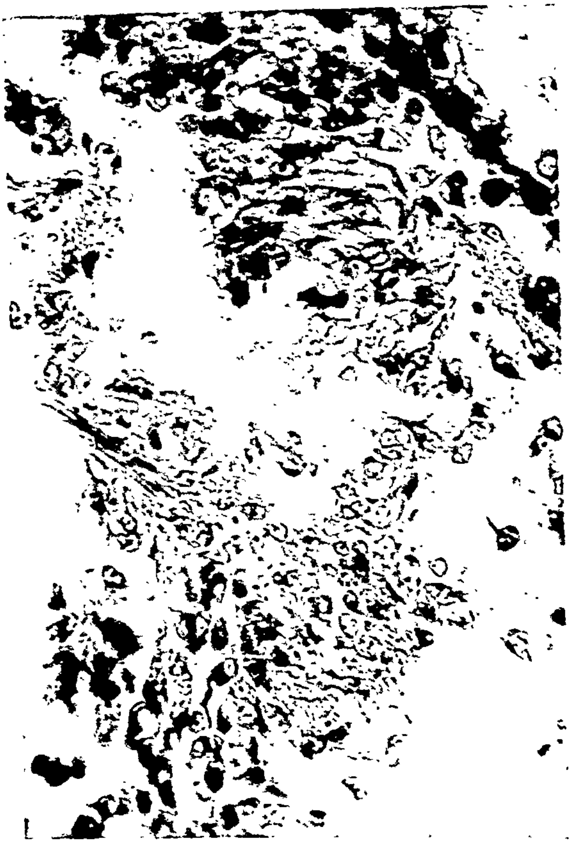


Fig. 7.—Bronchoscopic biopsy demonstrating undifferentiated carcinoma. Case 2. (X540.)

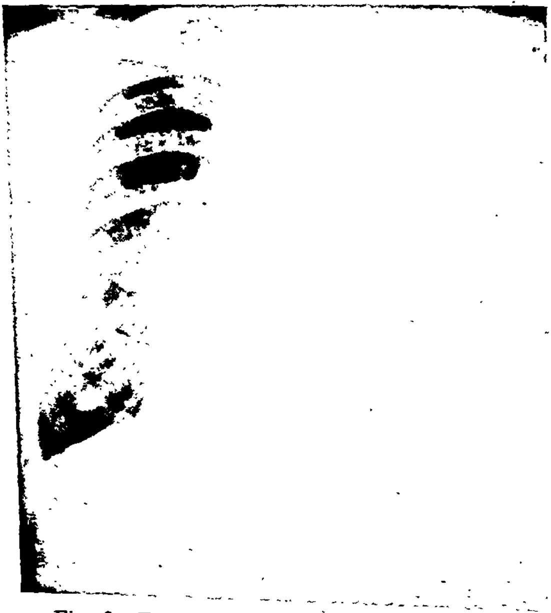


Fig. 8.—Extensive left pleural effusion. Case 3.

sarcoma; however, the cell patterns were not entirely comparable to the findings in the abdominal wall tumor.

The doses of radioisotopes used in this study have not produced evidences of toxicity in any patients. There have been no instances of radiation sickness, and no anemia or leucopenia has been observed to date. While the single dose employed in most patients was not large, one patient in this series (Case 10) was given several treatments and a fairly large amount of radioactivity. This patient was most refractory to this type of therapy. A very profuse, extremely bloody effusion was present which responded very slowly, so that over a period of seven weeks one injection of tagged serum albumin (5 mc.), three injections of I^{131} (totaling 55 mc.) and a single injection of Au^{198} (20 mc.) were given. In the following two months the patient was aspirated on two occasions for a total yield of 1,170 c.c., so that another dose of isotope therapy was decided upon. At this time 100 mc. of Au^{198} was employed, the largest dosage any patient had been given singly or cumulatively. No toxic manifestations have developed in this man.

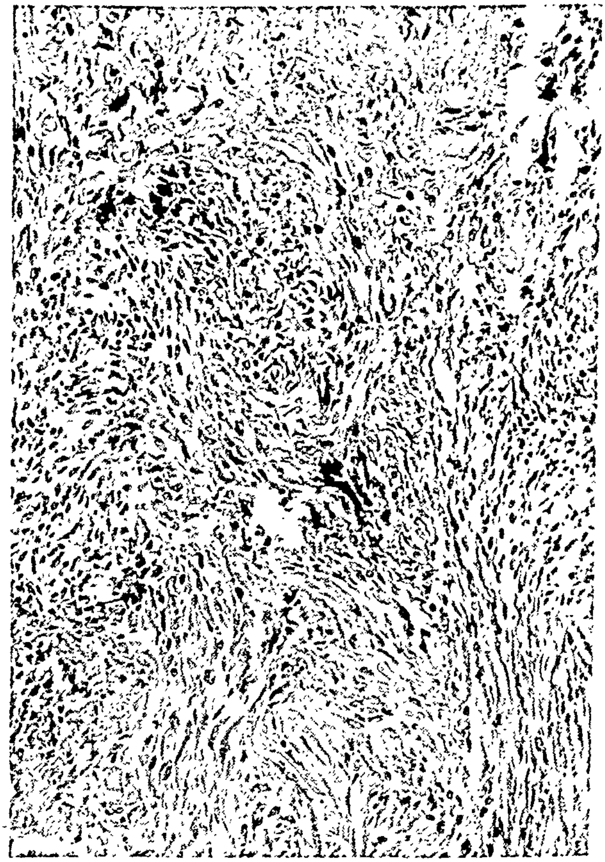


Fig. 9.—Micrographic picture of primary fibrosarcoma of abdominal wall in Case 3. (×135.)

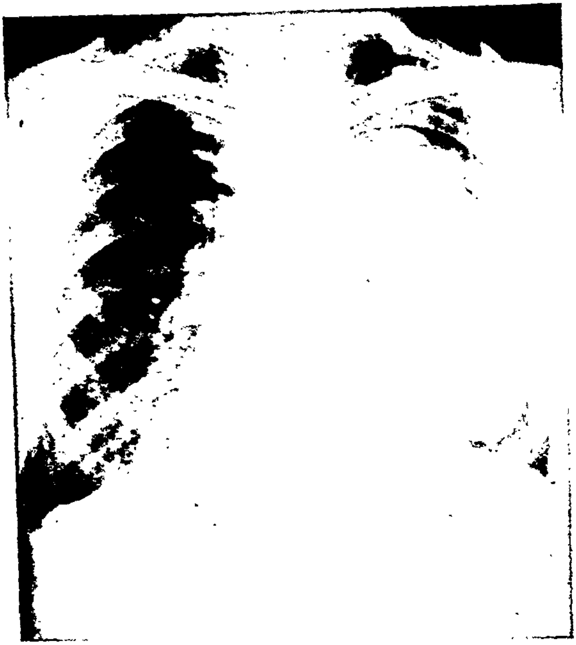


Fig. 10.—X-ray appearance of chest after removal of large amount of pleural fluid Case 3. Enormous left intrathoracic mass visible.

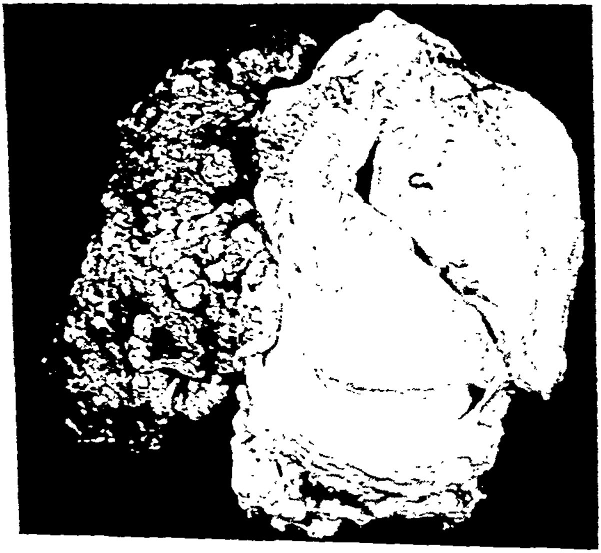


Fig. 11.—Autopsy specimen Case 3, revealing large left pulmonary metastases and numerous smaller right pulmonary metastases.

DISCUSSION

Because of the rapid disappearance of the I^{131} from the pleural space, it is difficult to explain why the beneficial response occurred. It seems unlikely that a beta ray tissue dosage large enough to cause severe destruction of tumor cells was delivered. However, there can be no doubt that fluid production was markedly retarded or stopped. Perhaps this can be accomplished with beta radiation dosage much smaller than that necessary to cause tissue destruction. In Case 9 assay of the residual radioactivity in a sample of pleural tissue taken at autopsy revealed an I^{131} concentration of 0.23 mc. per gram of tissue. Because of the indeterminate location of metastatic implants, it is impossible to calculate the distance between the pleural isotope and the lesion and, therefore, the amount of radiation received by the tissue cannot be reported in roentgens or roentgen equivalents. Additional experimental work is currently in progress in an attempt to determine the optimum radiation dosage and most suitable isotope for this type of therapy.

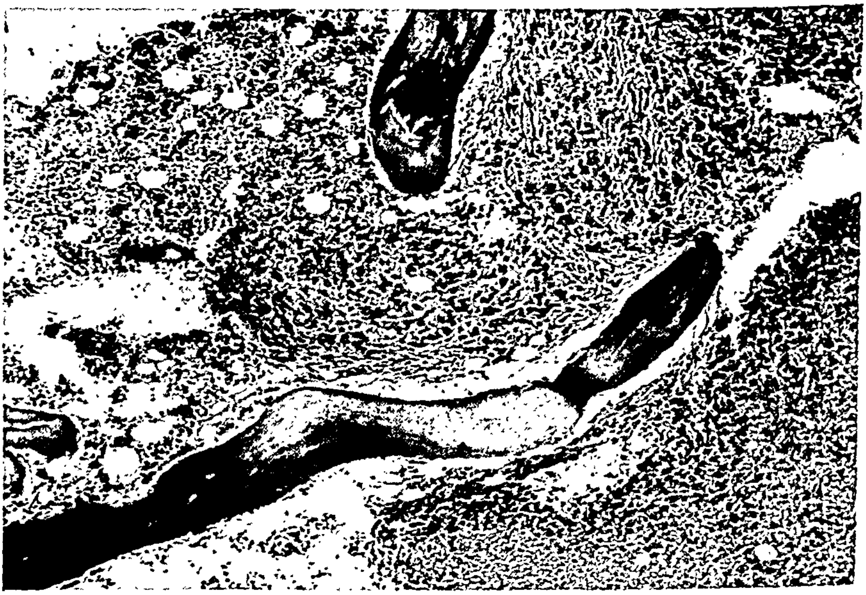


Fig. 12.—Bone marrow metastases found at autopsy, Case 3.

SUMMARY

1. A preliminary report on the first nineteen cases of pleural effusion due to metastatic carcinoma treated by the local injection of radioactive isotopes into the pleural space is presented.

2. Three of these patients died too soon after therapy to evaluate the results, and in three patients the formation of pleural fluid was definitely retarded but not entirely checked. In thirteen patients of this series with metastatic carcinoma the formation of pleural fluid was completely checked by the administration of radioactive iodine, iodine tagged albumin, or radioactive colloidal gold.

3. No toxic manifestations from the administration of the isotopes were noted in this series.

DISCUSSION

DR. J. KARL POPPE, Portland, Ore.—Dr. Kent and Dr. Moses are to be congratulated on this very careful work which represents a great deal of effort I am sure, because Dr. Julius Hessel and I undertook somewhat similar studies with insoluble redioactive ferric phosphate at the University of Oregon Medical School several years ago. This experimental work was confined to dogs and never reached the clinical stage because it seemed impossible to standardize the radioactivity of the ferric phosphate sufficiently to determine its minimal lethal or maximum effective dose. Due to the amphoteric nature of the phosphorus, the colloidal solution of ferric phosphate had a very vague end point on titration. It was very disturbing to discover that up to 50 per cent of the radioactivity of the original phosphorus was being lost in the supranatant fluid rather than being retained in the insoluble ferric phosphate suspension as was desired.

The original purpose in injecting this suspension of insoluble ferric phosphate into the pleural cavity was to irradiate the peribronchial nodes. The ferric phosphate was very easy to follow with iron stains on histologic sections, showing that the ferric phosphate was deposited in the peribronchial nodes. The radioactivity of these nodes was adequately demonstrated by autoradiographs. However, it seemed impossible to obtain a sufficient concentration of radioactivity in the nodes to destroy completely even a normal lymph node, not to speak of a node containing a large area of metastatic carcinoma. The normal morphology and histology of the node would be altered and the germinal centers eliminated, but some lymphatic tissue would always remain intact.

The use of radioactive material injected in the pleural cavity would seem to have definite advantages in maintaining a high concentration of irradiation around the pleural metastases if a completely insoluble substance is used which will not escape into the blood stream, liver, and bone'marrow.

DR. RALPH D. ALLEY, New Haven, Conn.—Dr. Kent has mentioned difficulty experienced in the total evacuation of serous effusions by ordinary thoracentesis technique, and I have a technical suggestion to offer based on some experience we have had in our chest service in New Haven.

We have developed an embellishment of the usual thoracentesis technique wherein a No. 14 needle is used in the usual manner through which is threaded a vinyl plastic tubing which just fits inside the diameter of the No. 14 needle, which can then be threaded down into the costophrenic sinus and total evacuation of any serous effusion carried out. I mention this because it may be helpful in his studies.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I think the authors of this paper are to be congratulated for discussion of something which seems to hold a lot of promise. About five years ago, working in Dr. John Lawrence's laboratory, we determined that simple solutions of radioactive phosphorus disappeared from the pleural cavity of dogs in a few hours. About two years ago at The Cedars of Lebanon Hospital, working with Dr. Henry Jaffe in the Department of Radiology, we began to use radioactive phosphorus in simple and colloidal solutions for pleural effusions due to carcinoma. About thirty patients have been treated. The same type of irregular result was noted by us as mentioned by Dr. Kent. Some patients seemed to get a marked dramatic response; others got no response. We then began to use radioactive gold (Au^{198}) for not only pleural effusion but in postoperative patients. So far, six such patients have been treated. One patient developed a bronchopleural fistula about four weeks after instillation of radioactive gold, and that was satisfactorily drained. Radioactive gold does not leave the pleural cavity as readily as does radioactive phosphorus. Also, it does not show itself in the field only. The radioactive energy is far more intense. Instead of Beta rays only, Gamma rays are emitted. The tissues take up the colloidal solution and fix it, so that although the liver is one of the points where fixation occurs, the tissues of the pleural cavity and mediastinum seem to be also a strong point of fixation of the colloidal gold.

As noted by Dr. Kent, very little is excreted in the urine. It seemed to us that this method might be useful in treating the patient in whom you have left behind some tumor in the mediastinum or pleura following resection. We encountered the same difficulties in calculating the tumor dose as mentioned by other speakers. The dosage usually was 22 mc. in about 30 c.c. of solution of the radioactive gold. In one patient with effusion with an inoperable carcinoma, we put 50 mc. of radioactive gold in the pleural cavity. So far we have seen no effect radiologically after three months, but the tendency to formation of fluid has been lost. This patient developed bone marrow depression; this may be seen at about six weeks after intrapleural instillation.

DR. MOSES (closing).—Dr. Kent and I would like to thank Dr. Poppe and Dr. Alley for their comments, and Dr. Alley for his suggestion as to methods of evacuating the pleura more completely. We would like to emphasize that we do not know why these isotopes exert this effect on the pleural effusion. Certainly the radiation effects are not sufficient to destroy the metastatic lesions. We raise the question that perhaps tissue dosages that are inadequate to destroy the carcinomatous implants but still exert some effect on them may cause sufficient damage to prevent the recurrent formation of pleural fluid.

As to the choice of isotope, we are quite certain now that although most of our cases receive ordinary I^{131}, this is not the optimum form of therapy, and we are quite enthusiastic about the value of colloidal gold and also of iodinated serum albumin. The colloidal gold, as Dr. Goldman indicated, is picked up by the lymph nodes and by the liver parenchyma, and we currently have in progress studies to attempt to determine just what dosage of colloidal gold and iodinated albumin will be most effective in eradicating experimental carcinoma and, in particular, how high the dosage can be raised without deleterious effect locally and on the liver and other vital structures.

IDENTIFICATION OF REGIONAL LYMPH NODES IN THE TREATMENT OF BRONCHIOGENIC CARCINOMA

JOSEPH A. WEINBERG, M.D.
LONG BEACH, CALIF.

CARCINOMA is essentially a local disease in its early manifestations. It tends to be confined to the region of its origin and to the lymphatics which receive lymph from the involved organ. Therefore, a prime consideration in the surgical treatment of cancer is the thorough removal of the regional lymphatics. The importance of this principle of treatment is recognized and practiced in most situations in which cancer occurs, but it has received little attention in the surgical treatment of cancer of the lung. In a personal communication published in Taylor and Nathanson's monograph on "Lymph Node Metastases" in 1942, Churchill and Adams stated "the surgery of carcinoma of the lung, so far as the problem of metastases is concerned, is comparable to the treatment of carcinoma of the breast by simple mastectomy." The authors of the monograph go on to say, "technical advances will probably make the operation safer and may permit removal of most of the nodes likely to harbor metastases. Although the prospect is discouraging because of the inaccessibility of the node metastases, and the frequency with which they occur, these are the avenues which are open to progress."[1] Although it is almost a decade since these statements appeared, little or no progress has been made in this direction. I have been unable to find in the literature more than casual remarks to the effect that the regional nodes should be removed in pulmonary resection for bronchiogenic cancer, except the excellent description of the nodes given by Tuttle and Womack in 1934,[2] and the reference to the removal of specific groups of nodes contained in Jones' article published in 1947.[3]

During the past two and one-half years I have been studying the arrangement of the regional lymphatics of the lung by means of vital staining during surgery.[4] This study, augmented by studies of the pattern of lymph node metastases in cases of advanced bronchiogenic cancer, and reviews of the detailed descriptions of the lymphatics of the thorax made by Rouviere[5] and Delamere, Poirier, and Cuneo,[6] has resulted in the establishment of a technique which permits the exposure and removal of the several groups of lymph nodes on each side of the thorax which are most likely to be affected by metastases.

From the Veterans Administration Hospital, Long Beach, Calif., and the University of California at Los Angeles Department of Surgery.

This study was supported in part by a grant from Wyeth Incorporated, 1600 Arch Street, Philadelphia 3, Pa.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, New Jersey, April 16-18, 1951.

The procedure of vital staining of the thoracic lymphatics consists of the injection of pontamine sky blue into the lung as soon as the hemithorax is opened and the diagnosis is established. The dye is seen in the regional nodes within a few minutes after the injection is made. In some instances the staining of the nodes is preceded by blue streaking along the lymphatic channels. The vital staining makes it possible to recognize the location of the various groups of nodes which receive lymph from the lung. While most of the thoracic nodes can be recognized by their anthracotic pigmentation, this is not always sufficient for easy identification because of the tendency of the dust particles to be concentrated in the more central portion of the nodes. This tendency, together with the obscuration by the unpigmented capsule of the nodes and the "camouflaging" resulting from scattered anthracotic pigmentation in the perinodal tissues, often make recognition of very small nodes or deeply placed nodes difficult or impossible. The dye does not stain carcinomatous tissue, and stained nodes which contain deposits of malignant cells reveal the uncolored cancerous deposits against the blue background.

Pontamine sky blue, which is used for the vital staining, has the advantage of being only slightly diffusible when it is injected into the tissues. It tends to remain in the area in which the injection is made unless it is picked up by the lymphatic circulation. Once it is deposited in a lymph node it tends to remain there. Hyaluronidase (Wydase*) is added to the dye solution to accelerate its uptake by the lymphatics. The dye has no toxic effects in more than one thousand times the quantity used clinically for vital staining as determined by animal experiments.[7]

The only deleterious effect we have seen is the occasional slight pigmentation of the skin which probably results from accidental injection of the dye into a blood vessel. In the several cases in which this occurred the skin assumed a cyanotic appearance which cleared up in several days to a few weeks. It has never been a troublesome complication.

The technique of the staining procedure is carried out in the following manner. Five cubic centimeters of a 4 per cent solution of pontamine sky blue in distilled water is mixed with thirty units of Wydase. Three to five cubic centimeters of the mixture is injected superficially and deeply in the circumference of the pulmonary tissue near the root of the lung. Some of the dye is also injected against and into the wall of the main bronchus. Nodes and lymphatic vessels are seen to be stained within one or two minutes after the injection is made, but the staining of the more distant nodes is usually not apparent until ten to twenty minutes have elapsed. Excursions of the lung induced by the anesthetist at this stage accelerate the movement of the dye toward the lymph nodes. It is well to wait ten minutes or so after the injection before proceeding with the dissection in order to allow the dye to reach the more distant nodes. In the meantime one examines the mediastinum to determine the gross extent of cancer invasion.

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*A product of Wyeth Incorporated, Philadelphia, Pa.

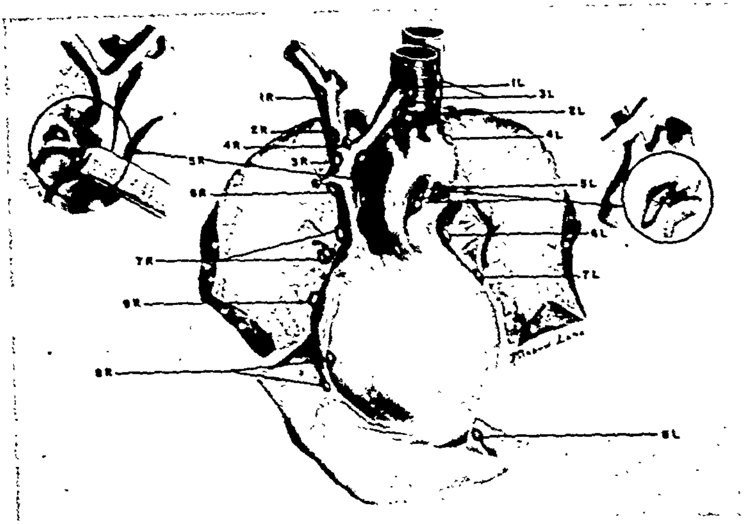

Fig. 2A.—Anterior view of regional lymph nodes of lungs and bronchi. *1R*-parasubclavian; *2R*—para-innominate; *3R*—paratracheal; *4R*—anterior mediastinal; *5R*—lower paratracheal; *6R*—azygos; *7R*—hilar; *8R*—nodes along phrenic nerve; *9R*—nodes of pulmonary ligament.

Left Side—*1L*—parasubclavian; *2L*—para-innominate; *3L*—paratracheal; *4L*—anterior mediastinal; *5L*—aortic window; *6L*—hilar; *7L*—nodes of pulmonary ligament; *8L*—nodes along phrenic nerve.

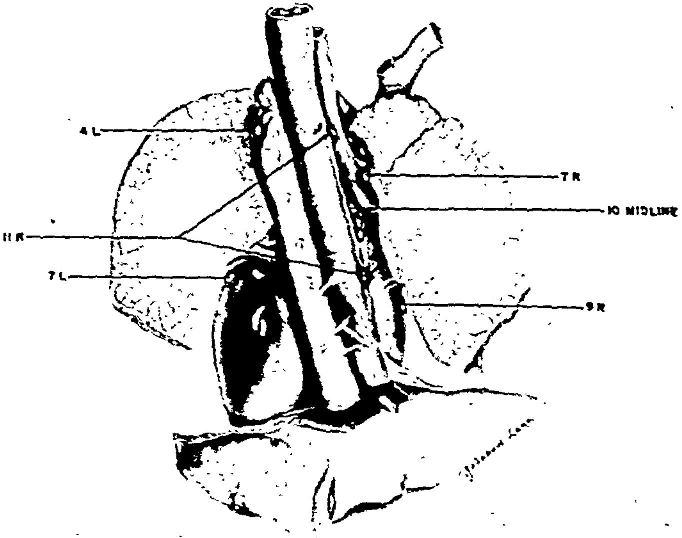

Fig. 2B.—Posterior view of regional lymph nodes of lungs and bronchi. *4L*—left anterior mediastinal; *7R*—right hilar; *7L*—nodes of left pulmonary ligament; *10*—midline, subcarinal; *11*—para-esophageal.

There has been no uniformity of success in staining the various groups of nodes. Sometimes an excellent injection of most of the groups of nodes is obtained; at other times only a few of the nodes take the stain. Many factors participate in determining the distribution of the dye. Probably the most important is the blockage of lymphatic pathways with carcinomatous tissue. Another is the presence of inflammatory changes which act as a barrier to the passage of the dye. Possibly a third is occasional departure from the usual injection technique. With improvement in the technique, successful staining of the nodes occurs with greater frequency. The procedure of vital staining has been used in seventeen cases of pneumonectomy, in five cases of lobectomy and in sixteen cases of simple exploration for malignancy, and in twenty cases of other types of surgical disease of the lung. At one time or another, all of the groups of regional nodes, which have been described by Ruviere[5] and by Delamere and his associates[6] in their complete monographs on the anatomy of the lymphatics, have been visualized. Vital staining of nodes has been seen in the opposite hemithorax in two cases in which death occurred within eleven days after surgery. This cross-staining occurred through the subcarinal and anterior mediastinal nodes. The nodes which have been stained also correspond closely to the pattern of the metastatic spread seen in individuals succumbing to bronchiogenic cancer.

Studies made on cadavers demonstrate that it is possible to remove most of the groups of regional nodes within the hemithorax with relative safety, and that in most instances it is possible to remove the nodes in continuity with the lung. We have altered our technique of pneumonectomy in several respects in order to accomplish this.

The approaches which permit the exposure of the several groups of nodes in each hemithorax will be described with the techniques for right and left radical pneumonectomy. Our preference is to attack the most peripheral groups first, and to carry the dissection toward the hilum without interruption. This procedure has the advantage of lessening the opportunity for embolic cancer cells. It also results in better exposure of the great vessels of the pedicle by first removing the lymph nodes and adventitia which obscure them. A lateral approach is made through the fifth interspace, as this gives a better exposure of all sides of the root of the lung than is obtainable with an anterior or posterior approach.

RIGHT RADICAL PNEUMONECTOMY

A longitudinal incision is made in the mediastinal pleura from the cupula to the azygos vein along the lateral margin of the superior vena cava. The nodes and adventitia on the under side of the subclavian vein (*1R*) and the nodes at the junction of the innominate veins (*2R*) are freed by sharp and gentle blunt dissection.

The dissection of the nodes and adventitia is continued downward en bloc to include the paratracheal (*3R*) para-esophageal (*3R*) and anterior mediastinal nodes (*4R*). Most of this dissection is facilitated by retracting the superior vena cava to the left.

When the azygos vein is reached, this vessel is divided between ligatures close to the superior vena cava, and the vena cava is retracted to the left to expose the important group of paratracheal nodes (*5R*) which lie in the space between the superior vena cava anteriorly, the trachea and the right pulmonary

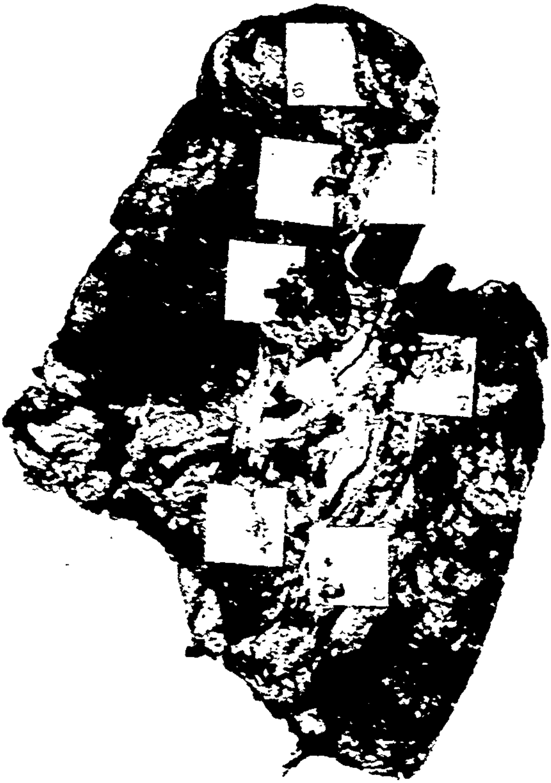

Fig. 3.—Right radical pneumonectomy with inclusion of regional lymph nodes. Arrow points to primary lesion in middle lobe bronchus. *1*—hilar nodes; *2*—nodes of pulmonary ligament and paraesophageal nodes; *3*—nodes along phrenic nerve; *4*—azygos nodes; *5*—paratracheal and paraesophageal; *6*—nodes along right subclavian and innominate veins; *7*—subcarinal nodes.

artery posteriorly, and the aortic arch to the left. The block dissection is continued downward toward the hilum to include this group and, at the same time, the nodes around the termination of the azygos vein (*6R*).

The right pulmonary artery which has been well exposed as a result of the preceding dissection is now doubly ligated proximally and distally, and is divided proximal to its branching. After dividing the mediastinal pleura over the anterior part of the pulmonary root, the hilar nodes (*7R*) and adventitia are thoroughly removed in this region and are left in continuity with the lung.

At this stage of the operation the exposed main bronchus is clamped to prevent the passage of secretions into the contralateral lung.

The phrenic nerve and the nodes along its course (*8R*) are removed with the vessels and areolar tissue accompanying the nerve. Although the direction of lymph flow is from the diaphragm toward the tracheobronchial nodes, the lymph vessels accompanying the nerve frequently show staining with the vital dye, and metastases have been seen in nodes along the phrenic nerve on several occasions. For these reasons it is advisable to remove these structures routinely.

The anterior and posterior leaves of the pulmonary ligament are divided close to the mediastinum in order to remove the nodes of the pulmonary ligament (*9R*) and the local paraesophageal nodes. In accomplishing this, the nodes and adventitia are maintained in continuity with the lung.

The inferior and superior pulmonary veins are ligated and divided close to the pericardium, care being exercised to dissect away the hilar nodes in this region and maintain their continuity with the lung. If the pulmonary veins are short and there is difficulty in dividing them, it is better to divide them within the pericardial sac rather than through their tributaries; otherwise one will have difficulty in including all of the hilar nodes with the resected specimen.

The lung is now retracted lateralward and upward in order to expose and remove the subcarinal nodes (10 midline). It is usually possible to remove all of these nodes well onto the under side of the left main bronchus. The nodes on the posterior aspect of the root of the lung are exposed by removing the mediastinal pleura from the posterior surface of the pedicle if this has not already been done. This will also expose the paraesophageal nodes (*11R*) lying above and below the level of the main bronchus. The esophageal nodes are not as likely to be involved as others, but, having seen them take up the dye, and having seen them involved with malignancy, we make it a practice to remove them together with the nodes on the posterior aspect of the pulmonary root.

The bronchus is now divided close to the trachea, and the stump is closed with interrupted sutures. Careful inspection is made for bleeding, particularly for bleeding from small vessels which have been interrupted during the removal of groups of lymph nodes. After it is ascertained that the field is dry, the intercostal incision is closed without drainage.

LEFT RADICAL PNEUMONECTOMY

The general plan of resection is the same as for right radical pneumonectomy, the differences being that there is a very important group of nodes within the "aortic window" to deal with on the left side, and there is no vessel which corresponds to the superior vena cava.

The mediastinal pleura is incised from the cupula to the upper limit of the pulmonary root. The left subclavian (*1L*) and innominate paravenous nodes (*2L*) are excised in the manner which has been described for the right side, and the dissection is continued toward the hilum, including the paratracheal (*3L*) and anterior mediastinal nodes (*4L*). A node is almost always found on the dome of the arch of the aorta near the origin of the left subclavian artery.

The nodes in the "aortic window," the space within the arch of the aorta bounded inferiorly by the left pulmonary artery, are thoroughly removed (*5L*). This group of nodes commonly receives metastases, particularly if the left upper lobe bronchus or main bronchus is the source of the malignancy. This dissection is performed by first dividing the ligamentum arteriosum between ligatures in order to obtain a free approach to the depth of the space. The nodes are removed together with those over the left pulmonary artery and bronchus. These nodes can usually be kept in continuity with the lung. One should carry the dissection within the arch of the aorta only to a distance which is considered safe for the control of bleeding from small vessels which may be interrupted. There is no apparent dividing line between these nodes and the right paratracheal nodes, and one must estimate the limit to which the dissection should be carried. There is probably no advantage in including right paratracheal

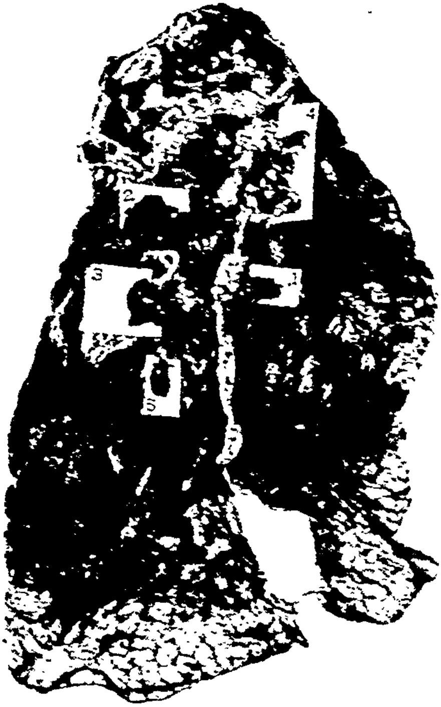

Fig. 4.—Left radical pneumonectomy with inclusion of regional lymph nodes. Primary lesion in apex of upper lobe. *1*—hilar nodes; *2*—nodes within aortic window; *3*—subcarinal nodes; *4*—paratracheal and anterior mediastinal nodes; *5*—nodes of pulmonary ligament and paraesophageal nodes. The phrenic nerve which lies to the right of the main bronchus is not labelled.

At this stage of the operation the exposed main bronchus is clamped to prevent the passage of secretions into the contralateral lung.

The phrenic nerve and the nodes along its course (*8R*) are removed with the vessels and areolar tissue accompanying the nerve. Although the direction of lymph flow is from the diaphragm toward the tracheobronchial nodes, the lymph vessels accompanying the nerve frequently show staining with the vital dye, and metastases have been seen in nodes along the phrenic nerve on several occasions. For these reasons it is advisable to remove these structures routinely.

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The general plan of resection is the same as for right radical pneumonectomy, the differences being that there is a very important group of nodes within the "aortic window" to deal with on the left side, and there is no vessel which corresponds to the superior vena cava.

The mediastinal pleura is incised from the cupula to the upper limit of the pulmonary root. The left subclavian (*1L*) and innominate paravenous nodes (*2L*) are excised in the manner which has been described for the right side, and the dissection is continued toward the hilum, including the paratracheal (*3L*) and anterior mediastinal nodes (*4L*). A node is almost always found on the dome of the arch of the aorta near the origin of the left subclavian artery.

The nodes in the "aortic window," the space within the arch of the aorta bounded inferiorly by the left pulmonary artery, are thoroughly removed (*5L*). This group of nodes commonly receives metastases, particularly if the left upper lobe bronchus or main bronchus is the source of the malignancy. This dissection is performed by first dividing the ligamentum arteriosum between ligatures in order to obtain a free approach to the depth of the space. The nodes are removed together with those over the left pulmonary artery and bronchus. These nodes can usually be kept in continuity with the lung. One should carry the dissection within the arch of the aorta only to a distance which is considered safe for the control of bleeding from small vessels which may be interrupted. There is no apparent dividing line between these nodes and the right paratracheal nodes, and one must estimate the limit to which the dissection should be carried. There is probably no advantage in including right paratracheal

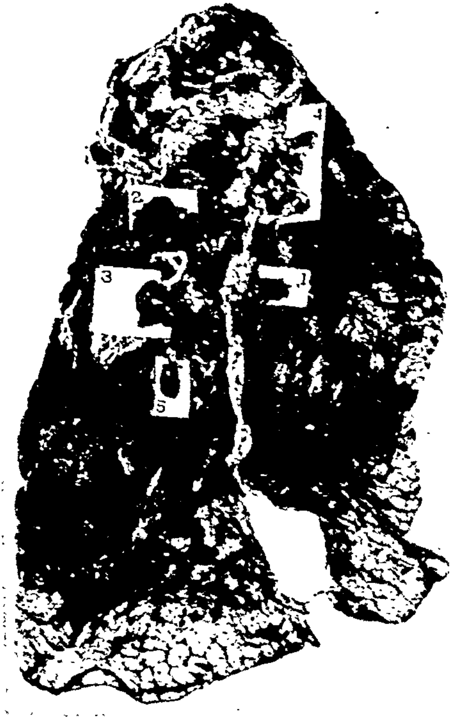

Fig. 4.—Left radical pneumonectomy with inclusion of regional lymph nodes. Primary lesion in apex of upper lobe. *1*—hilar nodes; *2*—nodes within aortic window; *3*—subcarinal nodes; *4*—paratracheal and anterior mediastinal nodes; *5*—nodes of pulmonary ligament and paraesophageal nodes. The phrenic nerve which lies to the right of the main bronchus is not labelled.

nodes, since there is no natural communication between them and the nodes of the left side. After completing the removal of the nodes in the "aortic window," the exposed left pulmonary artery can be seen to its origin from the main pulmonary artery.

The remainder of the resection is carried out in the same manner as on the right side.

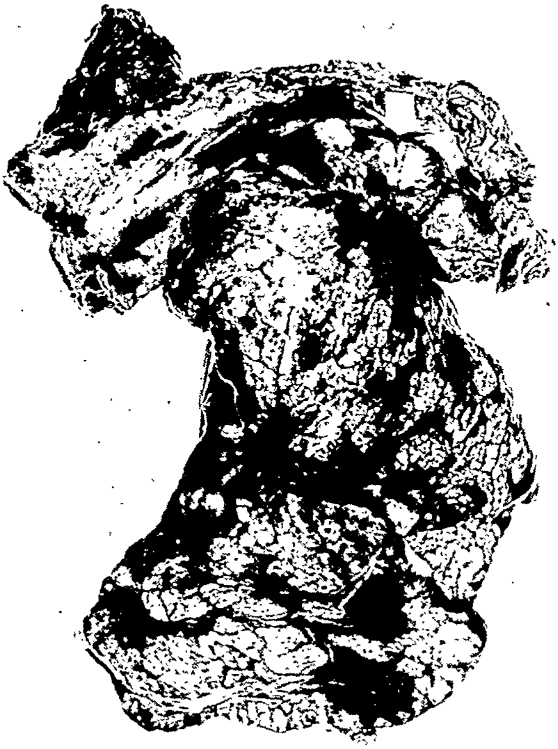

Fig. 5.—Cancer adherent to fourth rib, laterally. The third, fourth, and fifth ribs with parietal pleura, anterior and posterior intercostal nodes, and muscles overlying resected ribs have been removed en bloc with the resected lung and regional lymphatics.

RADICAL PNEUMONECTOMY WITH INCLUSION OF SEGMENT OF THORACIC WALL

The only nodes which remain to be considered are those of the parietal wall. While the parasternal nodes have taken up the dye in some instances, this has not been a frequent concurrence. The direction of the flow of lymph from the lung is not toward the nodes of the parietal wall of the thorax. We have limited the excision of the anterior and posterior chains of intercostal nodes to cases in which the carcinoma has become attached to the parietal pleura. When this situation is encountered, both chains are removed with a portion of the thoracic wall which is left attached to the lung. The excision is made en bloc, including at least one rib above and one rib below the site of attachment of the neoplasm. The parietal pleura, the muscles overlying the resected ribs, and the intercostal nodes in the segment of thoracic wall which is involved, are excised. There remains only subcutaneous tissue and skin over the area at the completion of the procedure.

While we have not yet performed dissections in the neck to remove the jugulo-subclavian nodes, this step is a logical one if nodes in the chain of communication from the lungs are to be removed as thoroughly as it is possible to do so. This might be performed several days before pneumonectomy.

There has been one fatality which can be ascribed to the extensiveness of the nodal dissection. This was in a patient with advanced cancer with metastatic involvement of most of the mediastinal nodes, in which all visible nodes were removed from under the superior vena cava. The patient died sixteen hours after operation, and at autopsy the hemithorax was found to be filled with clotted blood. No open vessels were discovered. It is assumed that bleeding from small vessels in the mediastinum caused the patient's death. Since this occurrence we routinely place pledgets of "gelfoam" soaked in thromboplastin in the space underlying the superior vena cava after removing the nodes from this area.

We have no statistics on longevity to report in these recent operations with vital staining as an adjunctive procedure. The emphasis which we have placed on extensive regional node resection is based solely on the premise that cancer, particularly cancer of the lung, spreads essentially by way of the lymphatics. It will take many years of experience with the procedure to permit a significant comparison of results of pneumonectomy with and without the inclusion of the several echelons of nodes which receive lymph from the lung.

SUMMARY

A method is described which is designed to facilitate the identification of regional lymph nodes of the lung for their removal during the operation of radical pneumonectomy for bronchiogenic carcinoma.

A solution of pontamine sky blue dye and hyaluronidase is injected into the pulmonary parenchyma as soon as the thorax is opened. The dye is taken up by the regional lymph nodes, imparting to them a blue color which makes them more easily distinguishable than they would be in their natural state. Many nodes which would remain obscured, even with acquired dust pigmentation, become readily identifiable after being colored by the dye.

The mapping of the nodes by vital staining, augmented by studies of the pattern of lymph node metastases in cases of advanced bronchiogenic cancer, has resulted in the establishment of a technique which permits the exposure and removal of the several groups of lymph nodes on each side of the thorax which are most likely to be affected by metastases.

This method is offered as a means of more nearly approaching the ideal of planned resection of lymph nodes, a phase of the operation which is of fundamental importance in the surgical treatment of bronchiogenic carcinoma.

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TEMPORARY UNILATERAL OCCLUSION OF THE PULMONARY ARTERY

A New Method of Determining Separate Lung Function and of Radiologic Examinations

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When elective surgery, which will impair the function of a lung is being considered, the functional state of the contralateral lung should always be taken into account. A simple estimate is often enough, particularly when the patient is young and the remaining lung exhibits no symptoms of disease. On the other hand, if the function of the remaining lung is doubtful, the examination should be rather more thorough, especially in cases where a certain amount of cardiorespiratory insufficiency already is present before the operation. In addition to the ordinary lung tests, it may sometimes be indicated to make a complete study of the circulation including cardiac catheterization.

However, no accurate estimate of the function of either lung can be obtained save by bronchospirometry. The latter method is, in its present state of development, capable of providing fairly accurate figures for the vital capacity of each lung and for its percentage participation in the absorption of oxygen. The bronchospirometric values recorded in air inhalation sometimes deviate slightly from those in oxygen inhalation. The former values are surely the most physiologic ones, but the difference may yield some information regarding the ratio between the volumes of residual air in the two lungs.

If bronchospirometry were combined with work, it seems likely that the maximal oxygen-absorbing power could be determined for each lung separately. Yet in the experiments so far carried out during moderate work (oxygen absorption four to five times normal) the percentage participation of each lung in the absorption of oxygen did not differ from conditions at rest (Björkman and Carlens[1]).

However, even after a study of the circulation, and having recourse to all conceivable information about the function of each lung, it will always be a matter of opinion whether or not a diseased lung can be removed in the individual case. In doubtful cases it would accordingly be highly desirable if it were possible before the operation temporarily to put the patient in a state which so far as possible resembled the functional condition he would be likely to be in after the operation. Attempts have been made to achieve this result by occluding the main bronchus to the diseased lung, or by letting it inhale

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nitrogen and the healthy lung oxygen while the bronchospirometric study is in progress (Jacobaeus and Bruce[2]). To prevent oxygen excretion from the lung which is breathing pure nitrogen, it has proved preferable to substitute artificial alveolar air for the nitrogen. The former gas mixture and the venous blood are in balance with one another, and no absorption or excretion can take place from the diseased lung. If the healthy lung thereby can be made to take care of the entire respiratory work without troubling the patient unduly, the test is a good one. Although these experiments can be made at rest, it is probably impossible to determine the respiratory reserve by carrying them out during physical work owing to the severe strain imposed on the patient. If the diseased lung functions comparatively well and its supply of oxygen is cut off under maintained blood circulation, the oxygen saturation of the mixed arterial blood will be reduced. Such admixture of venous blood from the occluded lung is most pronounced at first, but animal experiments have demonstrated that it is almost wholly compensated after about eight hours, since the circulation ceases almost completely in the nonrespiring lung (Euler and

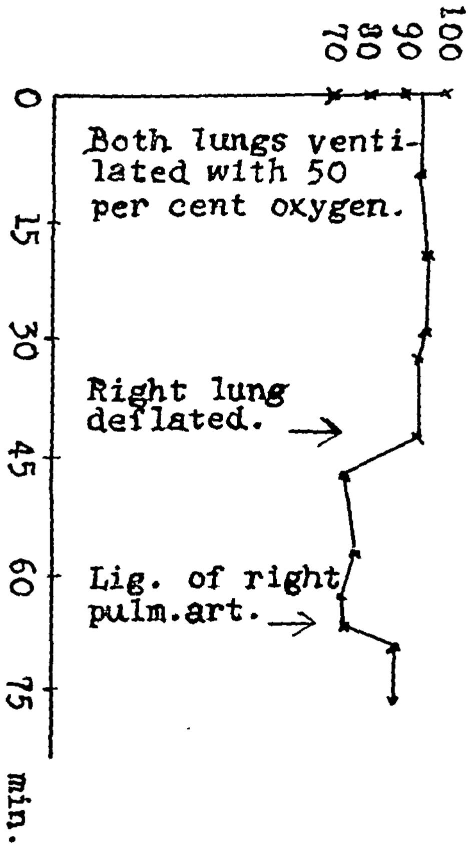

Fig. 1.—Oxygen saturation of the arterial blood during a right-sided pneumonectomy with pentothal-N₂O-curare anesthesia. Ventilation is maintained by spiropulsator through a double lumen tube to each lung separately.

Liljestrand,[3] Dirken and Heemstra,[4] Nisell[5]). However, there is much evidence to show that this reflex mechanism does not always operate in the same perfect manner in an abnormal lung in a middle-aged or elderly patient. Fig. 1 shows the arterial oxygen saturation as recorded by an oximeter, in a patient 67 years of age who was undergoing pneumonectomy under pentothal-nitrous oxide anesthesia. The patient's respiration was blocked by curare and maintained by a spiropulsator. A double-lumen tube enabled the lungs to be ventilated separately (Björk and Carlens[6]). Both lungs were ventilated at first, but as soon as the thoracic wall was opened up the healthy lung only was ventilated. At the very same moment the arterial oxygen saturation began to decline, although the inhaled gas consisted of 50 per cent oxygen: and it did not again begin to rise until the shunt had been bypassed by ligation of the pulmonary artery on the operated lung (Carlens and Finnerty[7]).

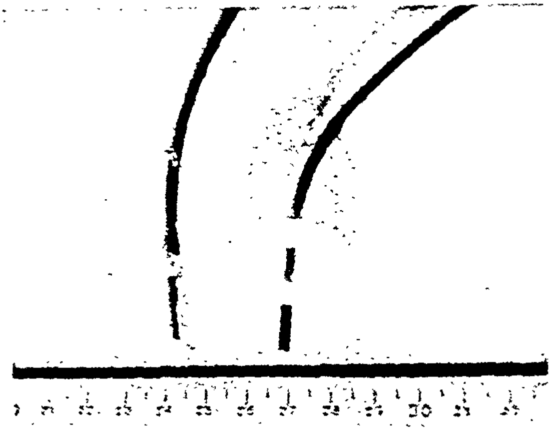

Fig. 2.—Photograph of cardiac catheter provided with cuff (inflated).

In some such cases, therefore, it seems likely that the patient will do well after extirpation of the diseased lung, though he cannot tolerate the effect when its oxygen supply is discontinued preoperatively. This behavior is due to the presence of a venous shunt in the latter case. Thus, theoretically, a stoppage of the function of the diseased lung by occlusion of its branch of the pulmonary artery would seem to be less trying to the patient and more in line with postoperative conditions when all the venous blood flows to the remaining, respiring lung. So far as we can find no such experiments have hitherto been made but, technically, the procedure should offer no serious difficulties if the method of heart catheterization is adopted.

METHODS

The opening in the tip of an ordinary Cournand cardiac catheter was filled in and a few centimeters proximally of it another one was made. Over it was placed an 8 mm. long, inflatable cuff of strong and thin latex rubber. When inflated the cuff assumed the shape of a short cylinder having a diameter of up to 3 cm.

The catheter was inserted in the usual manner in an exposed elbow vein or, in the preliminary experiments on dogs, in an exposed vein in the groin. Under fluoroscopic control the catheter could at will be made to enter the right or the left branch of the pulmonary artery. The rubber cuff was then slowly distended by injection of a 35 per cent solution of Umbradil through the catheter. This makes it easy to follow the distention and shape of the rubber balloon on the fluoroscopic screen. Consequently, the risk of excess pressure in the cuff is small and therefore damage to the vascular wall need not be feared.

The experiments on dogs were performed under barbiturate anesthesia, and 1 ml. of heparin was given before the catheterization to prevent clotting of the stagant blood distal to the balloon.

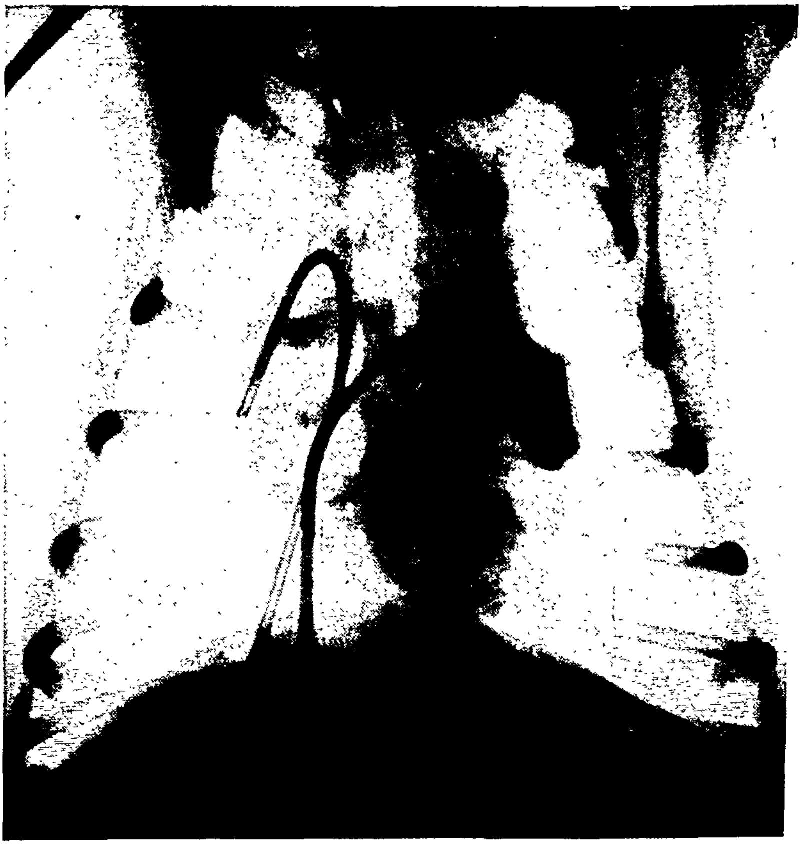

Fig. 3.—Roentgenogram of a dog with an inflated balloon in the left pulmonary artery and a conventional catheter in the right one. Both catheters inserted through the saphenous vein.

RESULTS

Ten experiments were performed on eight dogs, and in no case could we observe any complications following unilateral occlusion of the pulmonary artery. In one case one of the main branches was kept in a state of total occlusion for two hours, and in another it was impossible to find any electrocardiographic changes or other signs of complication despite very rapid and hard inflations.

How the pressure varies in the pulmonary artery has not yet been studied in detail, but in a few cases another catheter was introduced into the main trunk of the vessel, a slight increase in pressure being registered in immediate association with occlusion of one of the main branches.

In some instances the study was supplemented with cardioangiography. The contrast medium was injected into an exposed vein in the contralateral

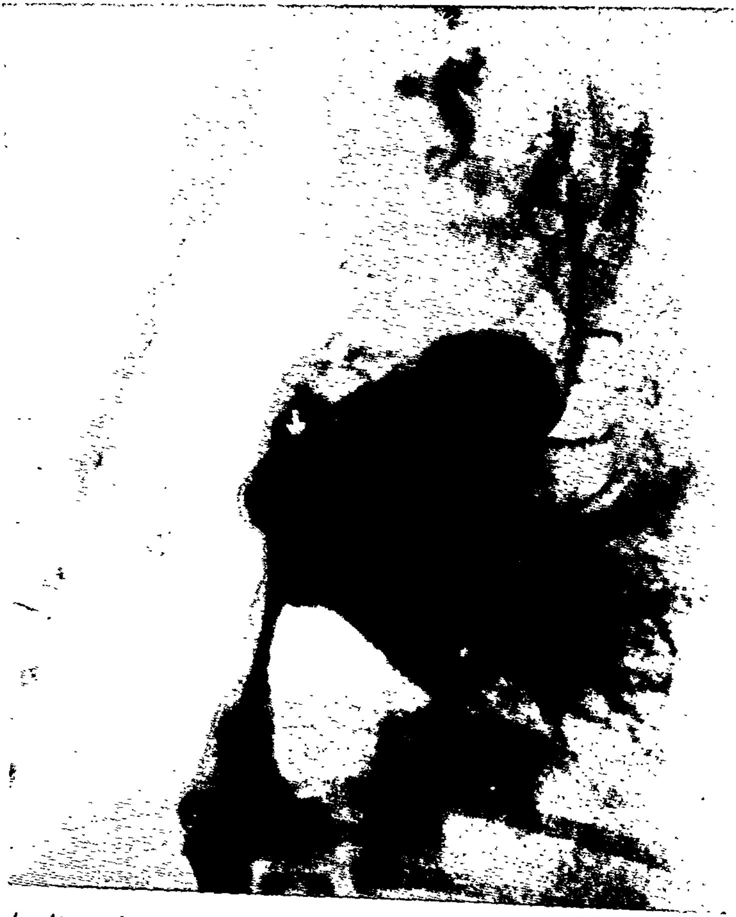

Fig. 4.—An angiocardiogram of a dog with inflated balloon in the right pulmonary artery. The catheter is inserted in the saphenous vein on one side and on the other side is a contrast medium (Umbradil) injected and has filled the pulmonary vessels on the left side only.

groin, and, as revealed by Fig. 4. the contralateral branches of the pulmonary artery were filled selectively. This is at least a rough indication that no blood passed beside the inflated cuff.

After a slight modification of the bronchospirometry catheter for use in human beings (Carlens[5]), we were able to perform bronchospirometry on two dogs in conjunction with unilateral occlusion of the pulmonary artery.

Fig. 5 shows such a registration when the right pulmonary artery was occluded by the inflatable cuff on the cardiac catheter. The ventilation was somewhat greater on the right side, but oxygen absorption had been taken over by the left lung. The right respirometric tracing was perfectly horizontal. As soon as the occluding balloon had been emptied by suction, the right respirometric curve rose as a sign of resumed oxygen absorption from the ipsilateral lung, and it at once declined when the balloon was reinflated. The total amount of oxygen absorbed per unit time remained constant whether or not the right branch of the pulmonary artery was occluded, demonstrating that

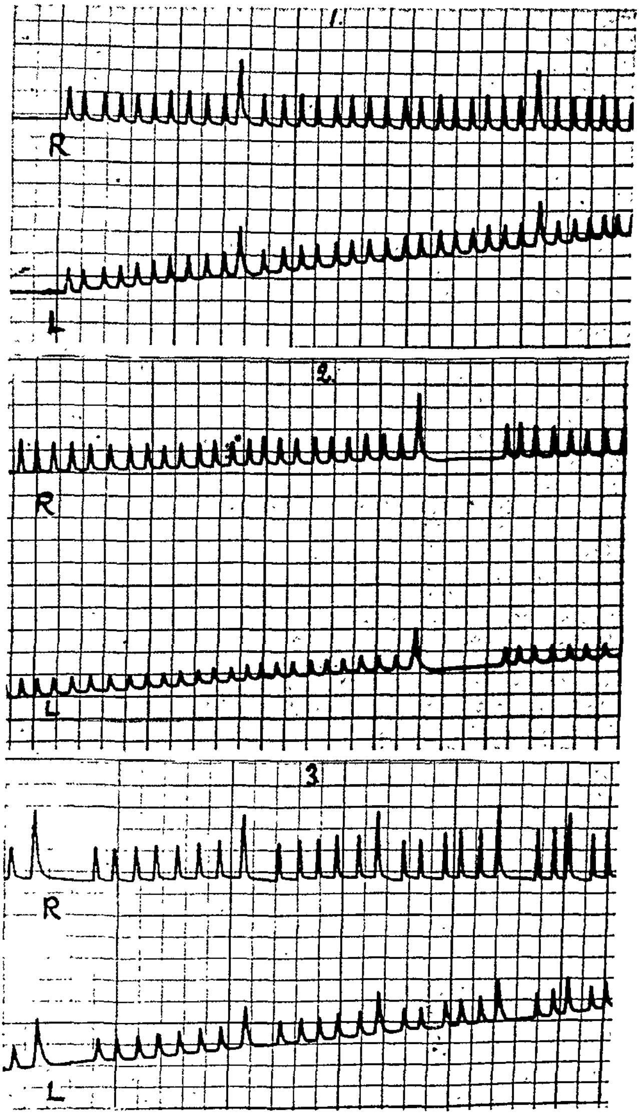

Fig. 5.—Bronchospirometric tracings in a dog with a cardiac catheter with inflatable cuff in the right pulmonary artery. *1,* Cuff inflated. No oxygen uptake in the right lung. *2,* Cuff deflated. Oxygen uptake in both lungs. *3,* Cuff reinflated. Oxygen uptake in left lung only.

the *nonoccluded* lung immediately took over the entire function. Such a bronchospirometric study should be a good check on the completeness of the fluoroscopically visualized occlusion.

No complications could, as noted, be observed, and all the dogs survived the operation without any visible disability. On two dogs the experiment was repeated with the same results after two weeks. One dog, killed three days after the experiment, was autopsied, but no macroscopical changes were observed in the occluded lung. Nor were any changes discovered by microscopical inspection of the vascular wall at the site of the inflated balloon.

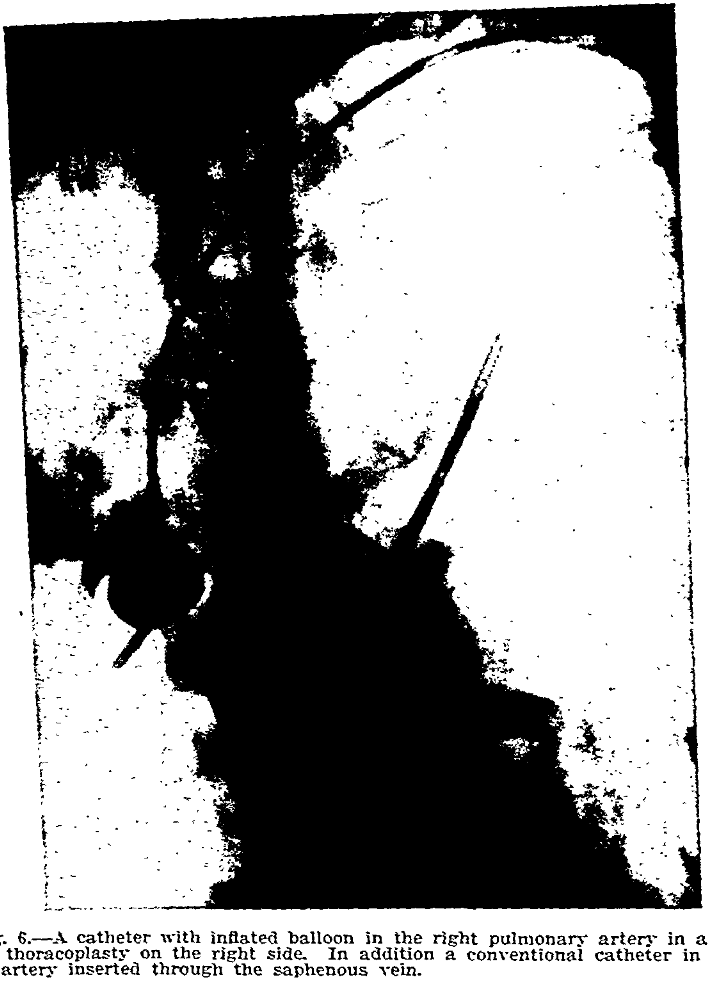

Fig. 6.—A catheter with inflated balloon in the right pulmonary artery in a patient who has had thoracoplasty on the right side. In addition a conventional catheter in the left pulmonary artery inserted through the saphenous vein.

EXPERIMENTS ON MAN

Since our animal experiments in no case exhibited any signs of complications, we considered it justified under the strictest precautions to try the method on three patients, who were considered for pneumonectomy.

Catheterization and occlusion of a main branch of the pulmonary artery were performed under local anesthesia after premedication with nembutal. The patients were given 3 ml. of a 5 per cent solution of heparin intravenously. The pulmonary artery was occluded for periods lasting up to twenty minutes. None of the patients experienced any subjective embarrassment from the chest. The rate and character of the pulse remained unchanged in occlusion, and so did the blood pressure in the brachial artery. In one case the mean pressure in the pulmonary artery, measured through a second catheter, was seen to rise from 17 to 22 cm. of water, but it regained its initial level after ten minutes. In one case no rise in pressure was recorded when the pulmonary artery was subjected to unilateral occlusion. In one case electrocardiogram tracings were recorded in three standard leads while the pulmonary artery was occluded repeatedly. There were no electrocardiogram alterations. In no case were there any complications following the experiment. None of the patients suffered any distress other than the sensations that usually accompany cardiac catheterization.

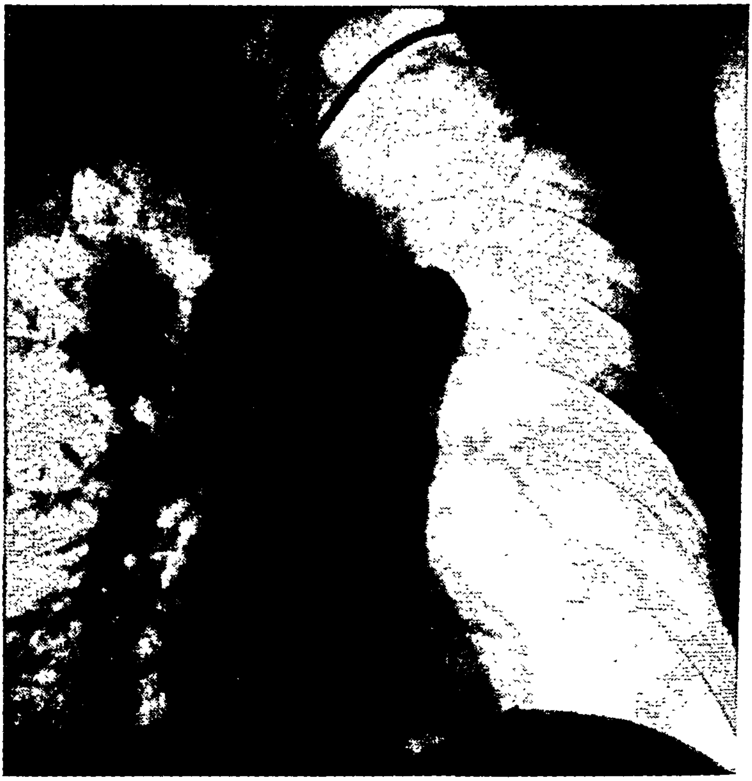

Fig. 7.—A roentgenogram of a patient with a catheter with inflated balloon in the left pulmonary artery. In addition contrast medium (Umbradil) has been injected through a conventional catheter in the superior vena cava and has filled the vessels on the right side only.

Thus, the preliminary results seem to indicate that it is possible in conscious human beings to occlude both the circulation in the pulmonary artery and the uptake of oxygen in one lung, and thereby approach as close as possible to the condition following a contemplated pneumonectomy. Under such conditions the circulation in the bronchial artery is not occluded, and consequently there is no need to fear lesions due to anoxia in the pulmonary tissues.

We intend, therefore, by this method to study the degree of dyspnea, the oxygen saturation of the arterial blood, the pressures, and resistances in the greater and lesser circulations at rest and during exercise.

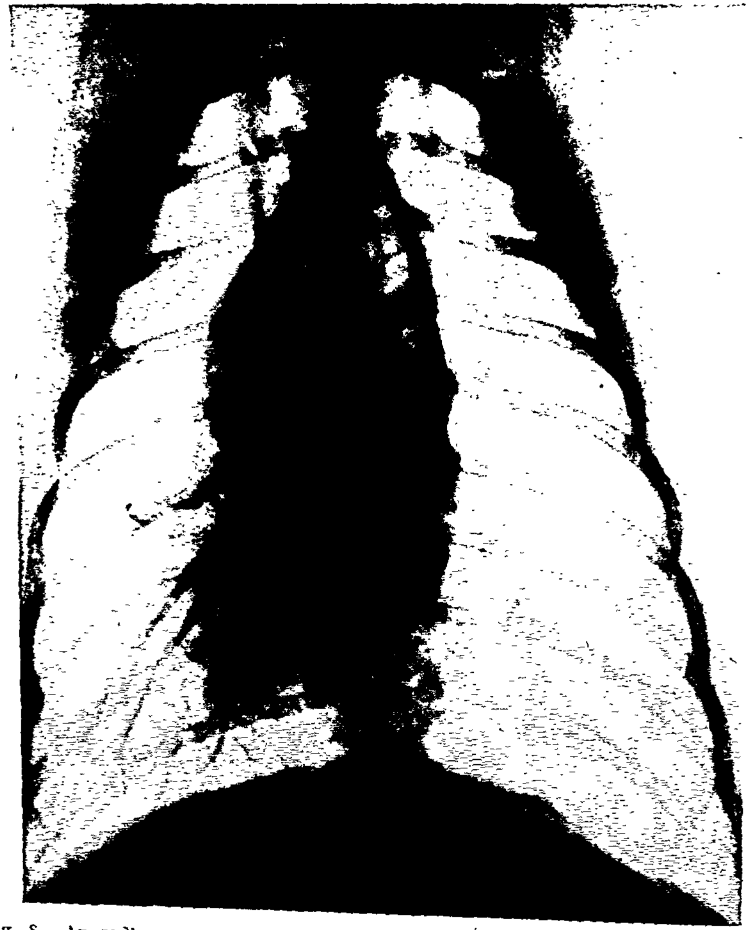

Fig. 8.—An ordinary roentgenogram of a dog with a double lumen catheter in the left pulmonary artery. The balloon is inflated and a small amount of a weak contrast solution is injected peripherally to it.

It is possible, moreover, that unilateral occlusion of the pulmonary artery may have radiological applications. By the methods used currently it may be impossible to visualize the pulmonary artery on one side in a lateral view, since the right and left sides are superimposed. By injection proximal to the occluding balloon the pulmonary vessels on one side can be filled with contrast medium. Contrast material may also be injected in the occluded pulmonary artery distal to the balloon, whereby it is unnecessary to use large amounts of highly concentrated contrast solution rather than small quantities of a solution which is isotonic with the tissues. There is no need of rapid serial exposures. The method is therefore likely to open up a means of studying in detail the vascular bed in the lungs. Experiments along these lines are at present in progress.

SUMMARY

In a preliminary experimental investigation on dogs, and in three cases on human beings, the pulmonary artery was occluded on one side by means of a cardiac catheter provided with an inflatable balloon. By this means the circulation in the pulmonary artery on one side and the oxygen uptake in the ipsilateral lung could be stopped. Thereby conditions were created which closely resembled the status following pneumonectomy, so that circulatory and respiratory changes resulting from extirpation of one lung could be foretold. No complications were observed.

It is possible that unilateral occlusion of the pulmonary artery may constitute a means for unilateral contrast radiography of the pulmonary vessels.

ADDENDUM

Since writing this paper, an article has been published by Charles Dotter and Daniel Lucas in *Am. J. Physiol. 164: 254, 1951. These authors have used a similar technique* and have done an experimental investigation on dogs. After occlusion of either the right or the left pulmonary artery they registered in every case a hypertension in the right ventricle and they did not continue the experiments more than three minutes because of grave symptoms of cor pulmonale in the dogs. Judging from the published photograph and figures, we feel certain that there must have been a total or subtotal occlusion of the main stem of the pulmonary artery in every case.

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OBSERVATIONS ON THE HEMODYNAMICS OF BRONCHIAL-PULMONARY VASCULAR COMMUNICATIONS

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IN THE course of certain experimental preparations in the dog intended to elucidate problems in pulmonary congestion, with particular reference to its effect on collateral respiration, rather unexpected and apparently paradoxical findings were encountered with regard to pressure changes in the pulmonary vascular tree. In brief, after complete occlusion of a lobar pulmonary artery and its corresponding veins, a progressive and striking elevation of endarterial pressure occurred in the occluded portion of the pulmonary vascular bed. This phenomenon seemed to us best explainable by the existence of a very active and immediate collateral circulation to the pulmonary vascular bed from the bronchial arteries at high systemic pressures. It is the purpose of this paper to describe further experiments employed in studying this mechanism, to present in detail the technical methods employed, to set forth the experimental data obtained, and to discuss the significance of these findings in relation to normal pulmonary hemodynamics.

Microscopic vascular communications between the bronchial and pulmonary arterial systems at the capillary level have long since been described in the normal lung.[1-4] After interruption of the pulmonary artery[5] and in various pathologic states these, and possibly newly developed channels, reach macroscopic proportions.[6, 7] All previous observations on such anastomoses were obtained by study of microscopic serial sections, or by injection and perfusion techniques[4, 8] The literature to our knowledge contains no reports of in vivo experiments designed to record endovascular pressure changes within an otherwise normal pulmonary lobe after occlusion of both arterial inflow and venous outflow.

MATERIALS AND METHODS

All experiments were performed on presumably healthy stock dogs weighing 13 to 28 kg. under sodium barbital anesthesia (300 mg. per kilogram), employing intravenous sodium pentothal for induction. Thoracotomy was done by resection of the left fifth or sixth rib under positive pressure respiration, which was discontinued at the time of closure of the wound. The technique for occlu-

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[**] James Hudson Brown Memorial Fellow—Surgery.

sion of the appropriate vessels and the manner of their catheterization underwent considerable evolution during the earlier experiments. These will be summarized in the next paragraph. The arrangement finally evolved for occlusion of the left lower lobe pulmonary artery and vein through the closed chest by a snare cannula (Fig. 1) about an indwelling pressure-recording catheter is depicted *for the artery in Fig. 2, and for the vein in Fig. 3. The left lower lobe was* selected for this technique, because its supplying pulmonary artery and vein are easily accessible, and the exposure not complicated by the presence of an accessory or subcardiac lobe, as is the case on the right. Furthermore, advantage could be taken of the anatomic fact that the right accessory lobe vein usually joins with the left lower lobe vein close to the auricle. This permitted the retrograde introduction of a venous pressure-recording cannula through the sacrificed accessory lobe vein into the left lower lobe vein. The pulmonary artery pressures were measured in four experiments with a water manometer, and in nine subsequent experiments with a Hathaway multi-channel electronic

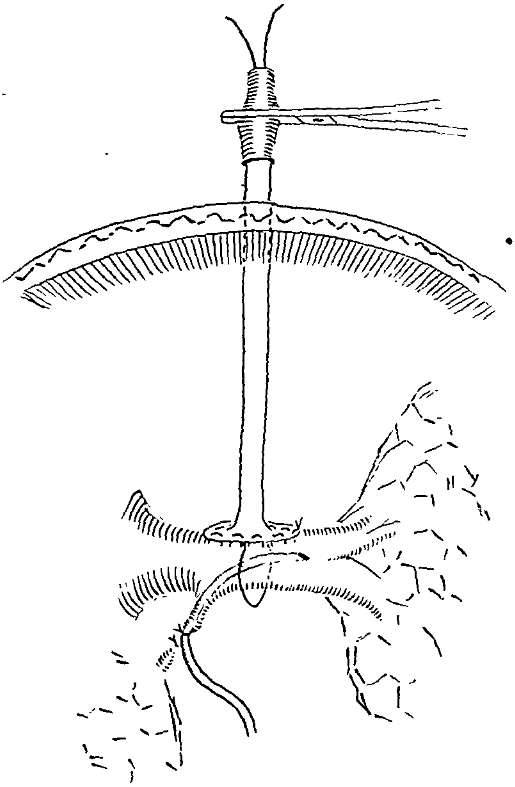

Fig. 1.—Diagram showing the polyethylene snare cannula sutured to the left lower lobe vein, with the snare loop passing through the cannula and around the vein. The end of the cannula is brought out through the thoracotomy wound with a tight closure of all layers around the cannula. To prevent ingress of air through the cannula, a rubber cuff surrounds its open end, which thus can be kept closed with a clamp except when manipulating the snare loop. The catheter for conducting intralobar pressures to the recording system is shown passing into the lower lobe via the right accessory lobe vein.

recorder.* Pulmonary vein pressures were recorded by the latter instrument in three experiments. Systemic blood pressure from a cannulated femoral artery was recorded by means of an ink-writing mercury manometer or with the electronic instrument. Intrapleural pressures were obtained by insertion through the left thoracic wall of an anchor-shaped glass cannula which was connected with an outlet of the electronic recorder. Continuous spirograms were recorded in three experiments employing an oxygen-filled Roth-Benedict spirometer with a one-way valve circuit and a CO_2 scrubber interposed connected to a tracheal cannula.

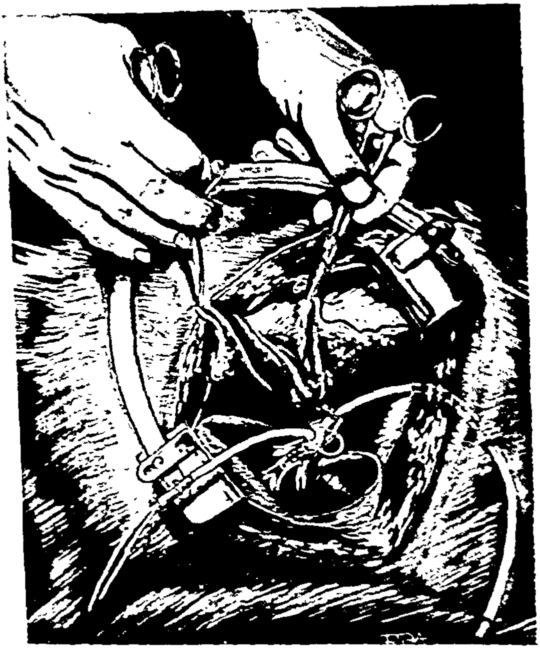

Fig. 2.—Showing the snare loop sutured to the left lower lobe artery, and the catheter passed via an upper lobe artery down into the lower lobe, past the point where the artery will be later occluded by pulling on the snare.

The principal variations in technique tested and subsequently discarded during the earlier experiments involved the manner of occluding the left lower lobe vessels. In the first experiment a double-lumen Cournand catheter with an inflatable balloon near its tip was introduced via the right external jugular vein during the thoracotomy, manually guided through the heart into the left lower lobe pulmonary artery, and the artery occluded by inflating the balloon. Because of the small lumen size of this catheter, an improvised double-lumen balloon-tipped catheter of polyethylene was employed in two subsequent experiments (No. 21 and 23). In the latter of these experiments a single lumen polyethylene catheter was also passed via the left auricular appendage into the left lower lobe pulmonary vein for recording its pressure, but it was found that dogs sion of the appropriate vessels and the manner of their catheterization underwent considerable evolution during the earlier experiments. These will be summarized in the next paragraph. The arrangement finally evolved for occlusion of the left lower lobe pulmonary artery and vein through the closed chest by a snare cannula (Fig. 1) about an indwelling pressure-recording catheter is depicted for the artery in Fig. 2, and for the vein in Fig. 3. The left lower lobe was selected for this technique, because its supplying pulmonary artery and vein are easily accessible, and the exposure not complicated by the presence of an accessory or subcardiac lobe, as is the case on the right. Furthermore, advantage could be taken of the anatomic fact that the right accessory lobe vein usually joins with the left lower lobe vein close to the auricle. This permitted the retrograde introduction of a venous pressure-recording cannula through the sacrificed accessory lobe vein into the left lower lobe vein. The pulmonary artery pressures were measured in four experiments with a water manometer, and in nine subsequent experiments with a Hathaway multi-channel electronic

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*Hathaway Instrument Company, Denver, Colo.

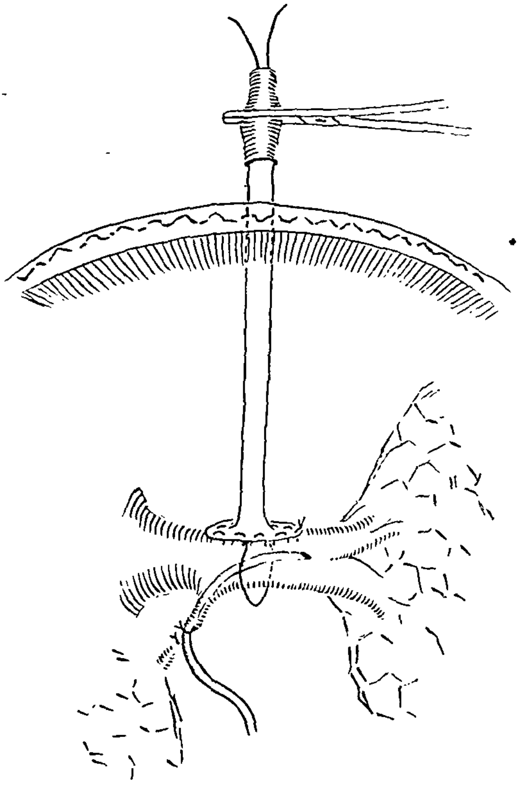

Fig. 1.—Diagram showing the polyethylene snare cannula sutured to the left lower lobe vein, with the snare loop passing through the cannula and around the vein. The end of the cannula is brought out through the thoracotomy wound with a tight closure of all layers around the cannula. To prevent ingress of air through the cannula, a rubber cuff surrounds its open end, which thus can be kept closed with a clamp except when manipulating the snare loop. The catheter for conducting intralobar pressures to the recording system is shown passing into the lower lobe via the right accessory lobe vein.

recorder.* Pulmonary vein pressures were recorded by the latter instrument in three experiments. Systemic blood pressure from a cannulated femoral artery was recorded by means of an ink-writing mercury manometer or with the electronic instrument. Intrapleural pressures were obtained by insertion through the left thoracic wall of an anchor-shaped glass cannula which was connected with an outlet of the electronic recorder. Continuous spirograms were recorded in three experiments employing an oxygen-filled Roth-Benedict spirometer with a one-way valve circuit and a CO_2 scrubber interposed connected to a tracheal cannula.

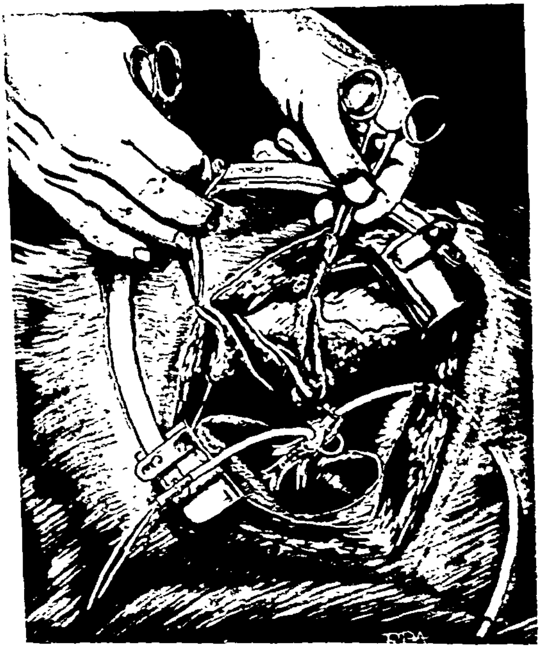

Fig. 2.—Showing the snare loop sutured to the left lower lobe artery, and the catheter passed via an upper lobe artery down into the lower lobe, past the point where the artery will be later occluded by pulling on the snare.

The principal variations in technique tested and subsequently discarded during the earlier experiments involved the manner of occluding the left lower lobe vessels. In the first experiment a double-lumen Cournand catheter with an inflatable balloon near its tip was introduced via the right external jugular vein during the thoracotomy, manually guided through the heart into the left lower lobe pulmonary artery, and the artery occluded by inflating the balloon. Because of the small lumen size of this catheter, an improvised double-lumen balloon-tipped catheter of polyethylene was employed in two subsequent experiments (No. 21 and 23). In the latter of these experiments a single lumen polyethylene catheter was also passed via the left auricular appendage into the left lower lobe pulmonary vein for recording its pressure, but it was found that dogs tolerate intracardiac catheters poorly for long periods of time. Therefore, starting with experiment 24, cardiac catheters were discarded. Next, after an intercostal thoracotomy approach, short balloon-tipped polyethylene catheters were introduced through a left upper lobe artery, threaded down into the left lower lobe artery, and the other end of the catheter was brought out through a stab wound in the chest wall. Sizes of catheters were chosen such that the outside diameter of the catheter would be about one-third the inside diameter of the vessel, and the inside diameter of the catheter would admit a 15- to 17-gauge needle. The tiny homemade latex balloons proved troublesome, however, there being a tendency for the balloon to extrude itself proximally when inflated. Therefore, the snare-cannula technique mentioned above was finally adopted for the lobar artery as well as the vein. This technique proved satisfactory, and was used in all subsequent experiments.

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*Hathaway Instrument Company, Denver, Colo.

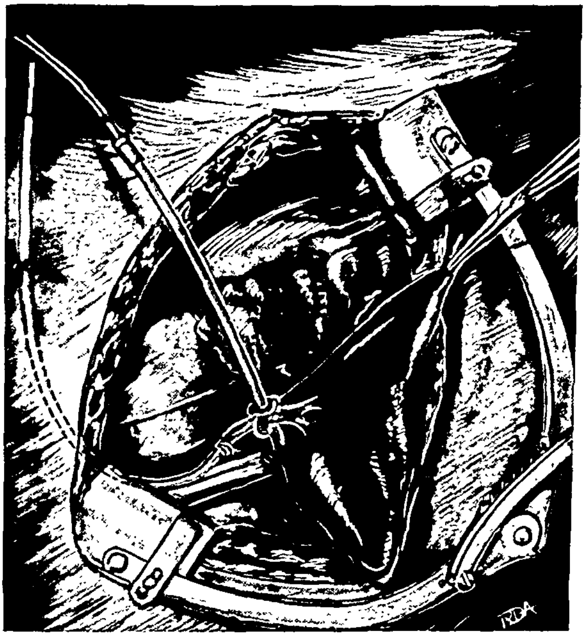

Fig. 3.—Showing the snare loop sutured to the left lower lobe vein at the hilus of the lobe, and the vein catheter passing via the right accessory lobe vein into the left lower lobe, beyond the point of later occlusion of the vein.

Two experiments (39 and 40) were chronic rather than acute. In these, simple ligation of the left main pulmonary artery was carried out five and six weeks, respectively, prior to the acute final pressure determinations, in order to study changes accompanying collateral expansion of the bronchial vascular system. At the time of the second thoracotomy in both animals, large, dilated, tortuous bronchial arteries were visible coursing over the hilar structures, mediastinum, and in the pulmonary ligament. The enlarged collateral vessels in the pulmonary ligament were sacrificed in the dissection exposing the right accessory lobe vein.

The effects of intravenous epinephrine, exsanguination, and subsequent intra-arterial retransfusion on intralobar pulmonary vascular pressures in the presence of occlusion of both lobar pulmonary artery and vein were also studied.

Following variable periods of hours during which the pressure studies were done, each animal was sacrificed and autopsied.

RESULTS

A. *Effect of Occlusion of Pulmonary Artery and Vein of the Left Lower Lobe in Normal Dogs.*—

Occlusion of a lobar vein alone caused an immediate moderate rise of intravascular pressure within the lobe, with a return to normal upon release of the vessel, as shown in Table I. The pressures reached exceeded the base line pressure by 3 to 18 mm. Hg.

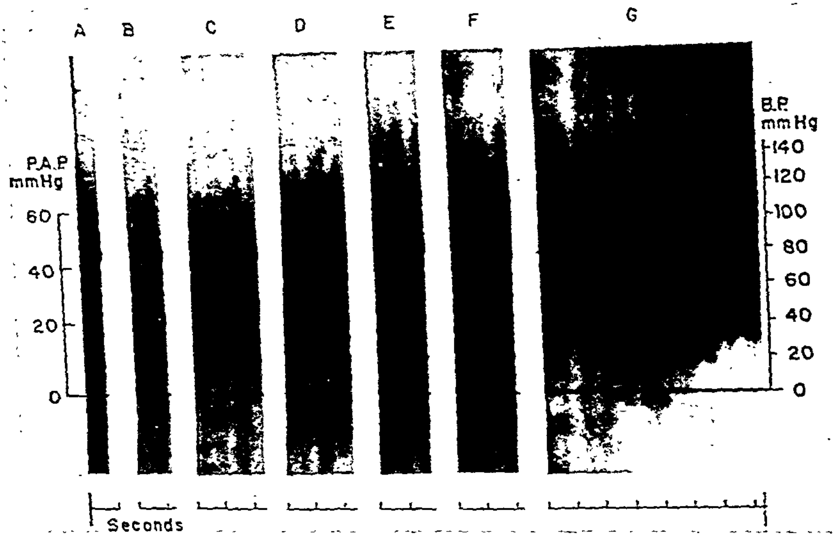

Fig. 4.—(Experiment 32.) Showing the effect of occlusion of the pulmonary vein and artery upon the pressure within the latter vessel distal to the point of occlusion. Upper tracing—systemic blood pressure (*B.P.*); lower tracing—pulmonary artery pressure within the obstructed area (*P.A.P.*). *A*, 12:33, base line. *B*, 12:35, after occlusion of pulmonary vein. *C*, 12:38, two minutes after occlusion of pulmonary artery, vein still occluded. *D*, 12:46, ten minutes after occlusion of pulmonary artery, vein still occluded. *E*, 12:47, eleven minutes after occlusion of pulmonary artery, vein still occluded. *F*, 12:51, fifteen minutes after occlusion of pulmonary artery, vein still occluded. *G*, 12:55, during release of pulmonary vein.

Occlusion of a lobar artery alone by intraluminal balloon or perivascular snare caused a prompt drop of pressure within the lobe, with disappearance of the pulse wave.

Occlusion of both lobar artery and vein, together in either sequence, was followed by a marked rise of pressure in the occluded segment to a level greatly exceeding the base line, pressures greater than 40 mm. Hg being reached in several instances as shown in Table I. A variable period of minutes was required for the maximum pressure to be reached (Figs. 4 and 5). From the Hathaway recorder tracings it was seen that at the height of pressure change a marked respiratory wave was still present. In those experiments wherein observation was prolonged for several hours, the elevated pressures were maintained at the peak level. Upon release of the lobar vein obstruction, pressures promptly fell to subnormal levels, following which release of the lobar artery occlusion resulted in a return to normal levels (Fig. 4). This sequence of events could be repeatedly demonstrated on the same dog, and was consistent in all thirteen experiments but one. At autopsy in this animal it was noted that venous occlusion was incomplete because a vein draining the apical segment of the lower lobe had not been included in the snare loop.

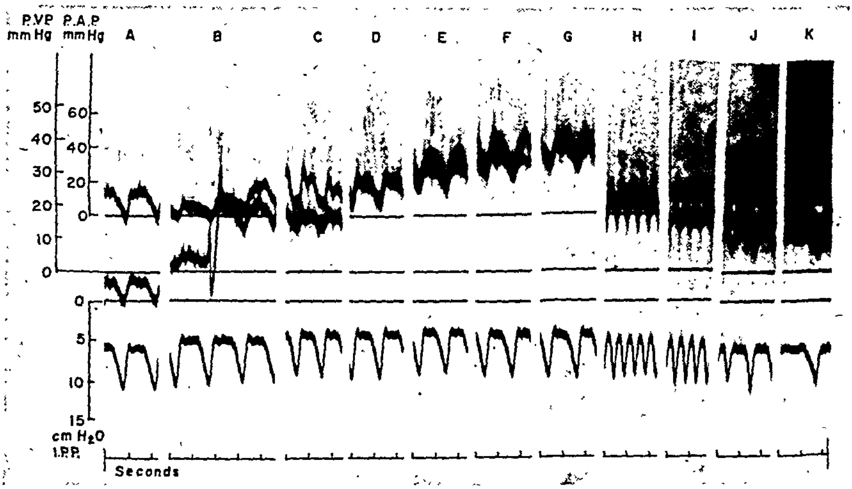

Fig. 5.—(Experiment 36.) Showing the rise in pulmonary artery and vein pressures produced by occlusion of both the artery and the vein. Also showing the effect upon the high pressure so produced within the lobe of systemic hypotension caused by bleeding. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural pressure (*I.P.P.*). *A*, 3:15, base line. *B*, 3:18, during occlusion of pulmonary vein. *C*, 3:19, after occlusion of pulmonary vein. *D*, 3:57, three minutes after occlusion of pulmonary artery, vein still occluded. *E*, 4:00, six minutes after occlusion of pulmonary artery, vein still occluded. *F*, 4:02, eight minutes after occlusion of pulmonary artery, vein still occluded. *G*, 4:13, nineteen minutes after occlusion of pulmonary artery, vein still occluded. *H*, 4:44, after 500 c.c. of systemic blood loss, with pulmonary artery and vein still occluded. Systemic blood pressure 80 mm. Hg, having dropped from an initial pressure of 120 mm. Hg. *I*, 4:47, after 700 c.c. blood loss, B P 50 mm. Hg. *J*, 4:58, blood pressure, 30 mm. Hg. *K*, 5:03, blood pressure, 25 mm. Hg.

In the three experiments wherein the lobar venous pressure was also recorded, this was found to parallel the changes in the lobar pulmonary artery pressure at a level lower by 5 to 10.0 mm. Hg.

B. *Effect of Occlusion of Pulmonary Artery and Vein of the Left Lower Lobe in Dogs With Expanded Bronchial Arterial System.*—

In the two dogs wherein an expanded bronchial arterial collateral system had been induced by previous ligation of the left pulmonary artery, the elevation in intralobar vascular pressure following occlusion of the left lower lobe artery and vein was more abrupt and reached a maximum in a shorter period of time than in the normal dog. (See Table I, Experiments Nos. 39 and 40). Whereas, the absolute peak pressure reached in Experiment 39 (45 mm. Hg) was not as impressive as that in Experiment 40 (75 mm. Hg) and actually fell short of that reached in four dogs with normal bronchial arterial systems, the rise observed, in terms relative to the systemic blood pressure of 80 mm. Hg, is significantly higher than that in the dogs with normal bronchial arterial systems and normal systemic pressures (Figs. 6 and 7).

C. *Effect of Systemic Blood Loss and Retransfusion.*—

To study the effect of these factors, rapid blood loss was produced via a cannula in the femoral artery, the blood thus withdrawn being later returned under sufficient pressure to cause a rapid rise of blood pressure.

With the lobar pulmonary artery pressure already at a maximum due to occlusion of the lobar artery and vein, systemic arterial hypotension produced by hemorrhage caused a reduction of intrapulmonary blood pressure commensurate with the drop of systemic pressure (Figs. 5 and 8).

Restoration of the animal's peripheral blood pressure by rapid intra-arterial transfusion caused a return of the pressure in the occluded lobar pulmonary artery to its previous high level.

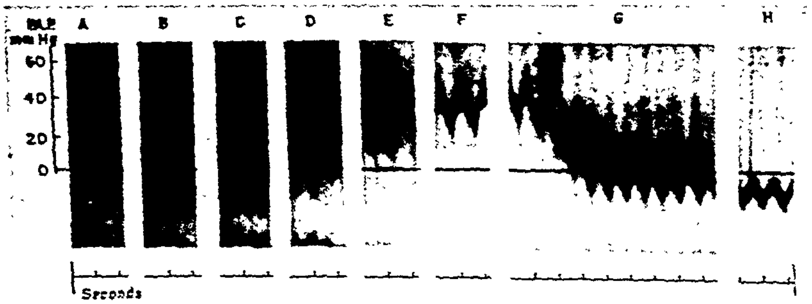

Fig. 6.—(Experiment 39.) Showing the rise in pulmonary artery pressure within the left lower lobe of a dog whose main left pulmonary artery had been ligated five weeks prior to the experiment. In this animal the systemic blood pressure was less than 80 mm. Hg throughout the course of the experiment, following the thoracotomy. The peak pulmonary artery pressures reached are high when considered in relation to the low systemic pressure. *A*, 1:10, base line. *B*, 1:17, one minute after occlusion of pulmonary artery. *C*, 1:20, immediately after occlusion of pulmonary vein, artery still occluded. *D*, 1:21. *E*, 1:22. *F*, 1:23. *G*, 1:24, during release of pulmonary vein. *H*, 1:27, after release of vein, pulmonary artery still occluded.

TABLE I. PRESSURE CHANGES FROM OCCLUSION OF PULMONARY ARTERY AND VEIN DISTAL TO OCCLUDED ARTERY

LOG NUMBER	PAP BASELINE (MM. HG)	AFTER PV OCCLUSION		AFTER PA AND PV OCCLUSION		SYSTEMIC BLOOD PRESSURE (SYSTOLIC MM. HG)
		MAXIMUM PAP (MM. HG)	TIME (MIN.)	MAXIMUM PAP (MM. HG)	TIME (MIN.)	
25	13	–	–	33	16	114
24	4	14	1	35	12	130
21	(5)	(15)	1	(10)	1	130
32	15	19	2	58	14	140
34	7	15	3	30	35	140
35	7	14	2	27	15	135
36	16	20	1	46	2	135
37	8	16	3	36	9	120
38	30	33	1	52	6	–
39	13	–	–	43	4	80
40	13	–	–	75	3	120
41	20	38	1	45	7	120
42	2	10	1	29	23	120

D. *Proof of Continual Influx of Bronchial Arterial Blood.*—

With the lobar pulmonary artery and vein occluded it was found that, if the pulmonary vein catheter cannulating the occluded lobar vessels be opened, blood would flow in a drip for an indefinite period of time from the end of the catheter. In order to rule out the possibility of passive outflow of blood from an engorged lobe, the external bleeding tip of the pulmonary vein catheter was elevated to a height of approximately 40 cm. above the pulmonary vein. Even against this imposed hydrostatic resistance, blood continued to drip at a rate of about six to eight drops per minute for prolonged periods.

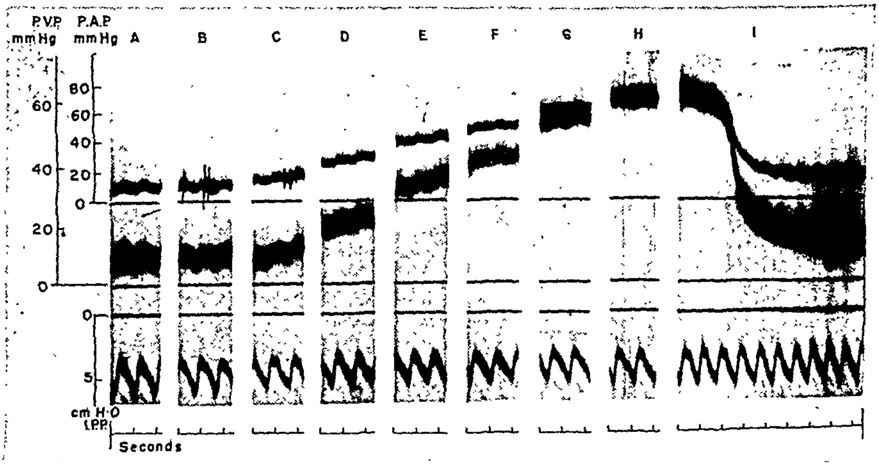

Fig. 7.—(Experiment 40.) Showing the rise in pulmonary artery and vein pressures of a dog whose main left pulmonary artery had been ligated six weeks prior to the experiment. *In this experiment the effect of occlusion of the pulmonary artery alone is not shown,* this vessel being occluded throughout the course. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural pressure (*I.P.P.*). *A,* 12:59, base line. *B,* 1:00, during occlusion of pulmonary vein. *C, D, E, F, selections from a continuous run immediately after vein occlusion. G,* one-half minute after occlusion of pulmonary vein. *H,* one minute after occlusion of pulmonary vein. *I,* 1:03, during release of pulmonary vein.

E. *Effect of Epinephrine.*—

With the lobar pulmonary artery pressure already at a maximum from occlusion of the lobar artery and vein, intravenous administration of epinephrine resulted in a still greater rise, occurring at the peak of systemic arterial pressure elevation, followed by a return to the pre-injection level paralleling the return of systemic pressure to normal.

F. *Other Effects of Occlusion of Pulmonary Artery and Vein of Left Lower Lobe.*—

I. *Systemic blood pressure:* There was no consistent effect upon systemic blood pressure due to occlusion of the vessels to a single lobe. In Experiment 30, which was one of the two dogs subjected to previous ligation of the left main pulmonary artery, there was a gradual decline of the systemic pressure over a twenty-five minute period following occlusion of both lobar vessels, from 100 mm. Hg to 50 mm. Hg. Upon release of the vessels there occurred a prompt return of the pressure to 80 mm. Hg. The fall might well be attributed to loss of blood into the lobe, and the rise to auto-transfusion upon reopening of the connections with the systemic circulation through the pulmonary vein and left heart.

II. *Respiratory effects:* After lobar vascular occlusion changes in the spirogram were seen inconstantly. When present these consisted of a gradual decrease of the tidal volume, usually with acceleration of the rate after variable time periods. This might be the result of the reflex described by Churchill and Cope,[9] who noted that increased pressure within the vessels of a single lung when produced by rapid introduction of excessive amounts of blood caused rapid shallow respirations.

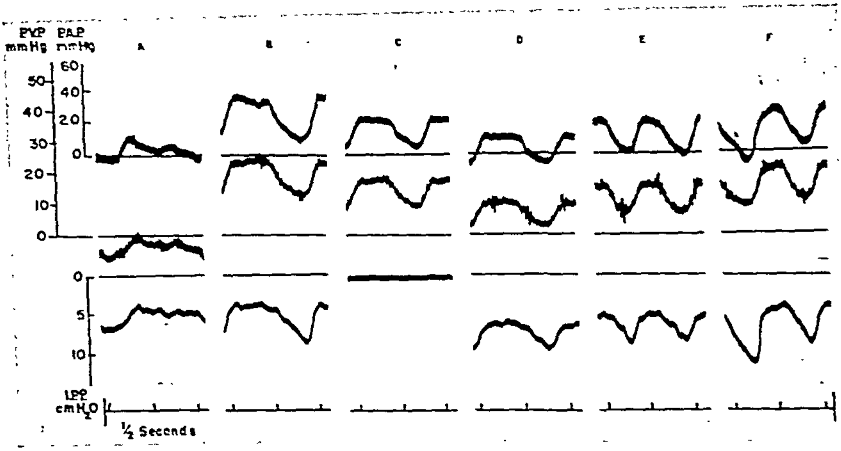

Fig. 8.—(Experiment 37.) Showing the effect upon intravascular pressure within the occluded lobe of systemic hypotension induced by hemorrhage and restoration of systemic pressure by retransfusion. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural artery and vein. *A*, 2:00, base line. *B*, 3:46, two minutes after occlusion of pulmonary artery and vein, blood pressure 120 mm. Hg. *C*, 3:55, after hemorrhage, blood pressure 75 mm. Hg. *D*, 4:00, after further hemorrhage, blood pressure 60 mm. Hg. *E*, 4:05, after retransfusion of blood, blood pressure 118 mm. Hg. *F*, 4:10, after further retransfusion, blood pressure 120 mm. Hg.

III. *Intrapleural pressure:* Pulmonary vascular congestion is known to produce changes in the dynamics of respiration which effectively alter the intrapleural pressure. Heyer and associates[10] induced congestion by administration of excessive amounts of fluid intravenously and found that there was increased amplitude of the intrapleural pressure swing with a consistent elevation of the expiratory pressure to values above atmospheric. In our experiments, with the congestion isolated to a single lobe, from occlusion of its particular artery and vein, consistent or significant effects on the intrapleural pressure were not seen.

IV. *Gross changes in the lobes:* The appearance of the left lower lobe varied depending on the period of time during which the vessels were allowed to remain occluded. Dense congestion was constant in some degree. If the period of occlusion was short, the color of the lobe was generally pink, whereas longer periods of congestion led to a dark, liver-like appearance, with the lobe enlarged and noncrepitant. Lung weight to body weight ratios were not computed. Gross evidence of pulmonary edema during the experiment, such as frothing of free fluid in the trachea, was not observed.

At the time of autopsy in each experiment the degree of occlusion of the vessels, whether by snare or balloon, was tested, and found to be complete in all instances, except in Experiment 21, where an apical segmental vein had not been included in the snare loop.

DISCUSSION

The rise in pressure observed within the pulmonary vascular bed of a lobe whose supplying pulmonary artery and vein had been obstructed is attributed to active congestion produced via the bronchial arteries. Although normally the bronchial arterial communications with the pulmonary circuit are by means of the intercapillary bed,[2] the intravascular pressures reached in the normal lobe after arterial and venous occlusion are far in excess of systemic (peripheral) capillary pressure. It is well recognized that the bronchial veins are poorly developed and are inadequate to accommodate more than one-third of the blood delivered by the bronchial arteries in unit time. This disproportion has been demonstrated anatomically and functionally by Berry and Daly[8] and by Bruner and Schmidt.[11] In the situation, then, of an obstructed pulmonary artery with impaired venous outflow, the so-called nutrient arteries of the lung become potentially a destructive force, raising the pulmonary vascular pressure far above the osmotic pressure of the blood plasma.

The phenomena noted above were in a sense predicted by Daly and his coworkers.[8] These workers, by an ingenious system of pumps, perfused the pulmonary artery and simultaneously the aorta or bronchial arteries of freshly sacrificed dogs, and then collected the blood from the left auricle and azygos vein. With pulmonary and aortic pressures both at normal limits, considerable blood passed from the bronchial to the pulmonary circuit. With the pulmonary artery pressure zero and aortic pressure normal, at least twice as much blood passed from the bronchial to the pulmonary circulation as from the bronchial circulation to the azygos vein. In the majority of their perfusion experiments an increase in aortic pressure raised the pulmonary arterial pressure as well as pulmonary venous flow.

Intravascular pressures observed in dogs with an abnormally "expanded" bronchial collateral circulation (produced by ligation of the left main pulmonary artery several weeks prior to the pressure studies) were elevated more rapidly and to proportionately higher levels following arterial and venous occlusion than in the normal dogs. The more rapid rise is to be related to expanded cross-sectional capacity and flow of the available bronchial arteries and a further development of extensive pre-capillary anastomoses between the bronchial and pulmonary arteries.[5] Thus the systemic pressure of the bronchial arteries is permitted to be more directly transmitted and felt in the pulmonary vascular bed. Increased bronchial collateral flow was demonstrated by our crude collection technique in the two dogs with previously ligated pulmonary arteries. An enormous increase in bronchial collateral flow following pulmonary artery ligation has been determined by the indirect Fick method and reported by Bloomer and associates.[12] It is unfortunately not known whether expansion of the bronchial venous system parallels that of the bronchial arterial system after pulmonary artery ligation. If not, exaggerated rises would in part be due to a progressive discrepancy between bronchial arterial inflow and bronchial venous outflow.

The above-described pressure changes in occluded pulmonary arteries of normal dogs support the existence of functional anastomoses between the bronchial and pulmonary arteries, an assumption previously shown to be true in the isolated perfused dog lung by Daly and associates, and by others[6, 7] in various chronic human pulmonary disease states.

The physiologic effects of the expanded bronchial vascular system in pathologic states of the lung has been postulated by Liebow and associates to be as follows: (1) The high pressure effects of the expanded bronchial arterial system, anastomosing with pulmonary arteries at the pre-capillary level serve as a shunt or hydrodynamic block to the inflow of unoxygenated pulmonary arterial blood to diseased portions of the lung, thus helping to maintain a more nearly normal mixed arterial oxygen saturation in the efferent pulmonary veins. (2) Abnormal bronchopulmonary communications with their high flow rate, draining mainly to the left heart by way of the pulmonary veins, may play a significant role in the development of left heart failure. (3) Conversely, blood shunted into the pulmonary arteries by way of bronchial anastomotic channels tends to increase the pressure within the lesser circulation, thus increasing the work of the right heart, and predisposing to cor pulmonale.

The functional significance of the communications which have been demonstrated in the normal animals above is speculative at the present time. The bronchial arteries are richly supplied with autonomic fibers.[13] It is possible that variations take place in the flow through these vessels, constituting a mechanism whereby deoxygenated pulmonary artery blood is shunted away from poorly ventilated alveoli. This is merely an application to the normal animal of a theory advanced by Liebow and associates to explain the shunting of blood which occurs in various pathologic states. Further field for speculation lies in the possible role of bronchopulmonary anastomoses in the pathogenesis of pulmonary edema due to increased filtration pressures.

Of greatest interest and importance is the role these communications may play in the fate of a pulmonary segment or lobe which has been deprived of its direct pulmonary arterial blood supply by an embolus or other occlusive pathology. If such a segment had also a high degree of functional venous obstruction, as from chronic passive congestion or mitral stenosis,[14] it is not improbable that bronchial collateral inflow at systemic pressures would produce a destructive imbalance, tending to favor the development of capillary engorgement, increased filtration, edema of the alveoli and interstitial tissues, diapedesis, and frank hemorrhage. This may be the mechanism of hemorrhagic infarction.

SUMMARY

When the pulmonary artery and vein of a lobe in the dog's lung are both occluded in an otherwise intact chest, the vascular pressures within the lobe

rise to abnormally high levels. This in vivo evidence supports the existence of functional communications between the bronchial and pulmonary arteries within the normal lung.

The possible significance of these communications is discussed.

We are indebted to Professor Averill A. Liebow, Department of Pathology, Yale University School of Medicine, for many valuable suggestions, and Mr. Armand Negri, Department of Surgery, Yale School of Medicine, for his technical assistance.

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Erratum

In the paper by Winfield O. Kelley, M.D., and David V. Pecora, M.D., Norwich, Conn., entitled "Revision Thoracoplasty" (J. Thoracic Surg. 22: 295, 1951), on page 295 in the first paragraph on line 19, the sentence "This method was successful in converting the sputum of 4.35 per cent of twenty-three cases" should read "This method was unsuccessful in converting the sputum of 4.35 per cent of twenty-three cases."

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Original Communications

PNEUMONECTOMY FOR SEVERE IRRADIATION DAMAGE OF THE LUNG

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ALTHOUGH thirty years have passed since Groover, Christie, and Merritt[1] first called attention to the syndrome of lung damage as a result of exposure to ionizing irradiation, no progress has been made in the treatment of this disease. In fact, a review of the literature reveals a virtually complete lack of consideration of this phase of the problem despite extensive preoccupation with the clinical and pathologic aspects of the syndrome. Since the first successful pneumonectomy for carcinoma was done in 1933,[2] vast progress has been made in thoracic surgery, and many conditions previously regarded as incurable have yielded to the surgical attack. When a patient with lung damage due to roentgen irradiation presented herself to us in 1947, it appeared logical to explore the question of whether this might not be one more condition amenable to successful correction by surgery.

Many case reports and experimental studies have been published since the original identification of the lesion, and this extensive literature has been reviewed by Desjardins,[3] Engelstad,[4] and more recently by Warren.[5] While a comprehensive review of the literature is, therefore, unnecessary at this time, a summary of the accumulated knowledge of the effect of irradiation on the lung is in order.

EFFECT OF IONIZING IRRADIATION ON THE LUNGS OF EXPERIMENTAL ANIMALS

Of the many experimental studies, only three will be considered here because they are representative and because the work to be discussed is of outstanding significance.

Lüdin and Werthemann's work,[6] although carried out on small numbers of animals, produced significant results. Their first series of experiments consisted of five rabbits given 500 r. of roentgen irradiation measured in air to alternate anterior and posterior chest fields measuring 5 by 5 centimeters. One treatment

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Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

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was given weekly until the animals died of the effects. From seventeen to forty-three treatments were administered. The changes produced consisted of increase in consistency of the lungs and production of pleural adhesions. Some parts of the lungs showed diffuse pneumonic infiltrations, others showed multiple small, firm, nodular foci of increased density. Many of the bronchi contained thick pus which was sterile on culture. In the least affected lungs the microscopic picture was that of patchy areas of atelectasis, and scattered alveoli with thickened walls. The alveolar capillaries were congested, and scattered alveoli contained edema fluid. In some alveoli degenerative changes in the lining cells were observed. The mildest changes in the bronchi consisted of hypersecretion of mucus and degeneration of the epithelium. In the severely affected areas purulent bronchitis, bronchiolitis, and bronchopneumonia occurred. This was accompanied by extensive destruction of alveolar epithelium and regeneration of the bronchial epithelium, as evidenced by the presence of scattered mitoses. The second series consisted of six rabbits which were given the same amount of irradiation, but the authors used 12 by 13 centimeter fields. This increased the amount of irradiation reaching the lung by an increase in the scattered irradiation. As a result the animals succumbed much earlier than those of the first series, so that only fourteen to fifteen weekly treatments were given. The grossly visible changes in these lungs were much more marked. Usually all parts of the lungs were affected, and individual foci could not be distinguished. The bronchitic, bronchiolitic, and bronchopneumonic processes observed in the more heavily irradiated animals of the first series were again seen. In addition, all the animals showed epithelial proliferations of varying degree. The bronchial epithelium became tall and stratified, and hypersecretion of mucin was marked. The most striking epithelial changes were seen in the peribronchial lung parenchyma. Alveolar structures lined by cuboidal epithelium are described and photographed, and are interpreted as arising from the bronchial epithelium. These authors believed these structures to result from infiltration of peribronchial tissue by proliferating bronchial epithelium which is thus regarded as showing autonomous growth characteristics. A study of the photomicrographs, however, suggests that these structures might represent peribronchial alveoli whose epithelium is very hyperplastic. Nevertheless, the concept that neoplasia might be induced by irradiation cannot be completely rejected. The frequent occurrence of bronchiogenic carcinoma in the Schneeberg miners is well known, and these growths may be instances of neoplasms induced by ionizing rays.

Engelstad's experiments[4] are the most extensive on record. One hundred sixteen rabbits were examined of which one hundred one had received roentgen irradiation to the chest, while fifteen served as controls. It was shown that different types of irradiation did not produce qualitative or quantitative differences in the effect on the lungs, provided that the total tissue dose and intensity of irradiation remained constant. Total dose, and the time factor, i.e., the intensity of irradiation, played a marked role in determining the severity of the reaction which became less with fractionation and protraction of a given dose of irradiation. The quantities of irradiation used in these experiments were based on the skin changes produced. Three types of irradiation were employed: subepidermicidal doses which provoked slight or no skin reactions, epidermicidal doses which produced an epidermitis lasting up to six weeks, and dangerous or lethal doses which caused severe skin reactions in all animals and led to death within a short time in many. The total dose of irradiation was given in the course of one day or in a few days, at most twelve.

After subepidermicidal doses lung changes were slight and recovery virtually complete within two months, leaving in its wake a mild fibrosis. Epidermicidal doses caused a rather severe and typical reaction which could be divided into four stages. The initial stage coincided in time with the early erythema of the skin, and consisted of degeneration of lymph follicles, hypersecretion of bronchial mucus, and hyperemia and leucocytic infiltration. The second stage, a latent period, lasted two to three weeks. After this the main reaction occurred. It was characterized by degenerative changes in the bronchial epithelium and in the pulmonary stroma and by various degrees of acute inflammation in the form of peribronchial, perivascular, and bronchopneumonic infiltrations. The bronchial cartilage proved to be relatively radioresistant. Alveolar macrophages, occasionally bizarre and multinucleated, were common. The last stage of the reaction began after the subsidence of the main reaction, usually after one to two months. Regenerative and reparative processes dominated this phase. Its main features were proliferation of connective tissue, sclerosis, and proliferative changes in the bronchial epithelium, and more rarely in the alveolar epithelium. These processes were not complete at the end of six months.

After dangerous or lethal doses the reaction differed from the above only in being more severe and in that the sclerosing processes of the reparative stage were more exuberant. Ossification of portions of the lungs was common. Thus Engelstad demonstrated a direct relationship between the amount of irradiation and its effect on the lung. He showed that small doses produce only mild or insignificant changes, and that the significant reaction caused by larger doses consists of an early acute inflammatory reaction and a prolonged late sclerosing process.

Warren and Gates[7] contributed significantly to our understanding of the pathology of the experimental radiation reaction by an exact histologic analysis of the mild early effects of irradiation on the lungs of rats, rabbits, dogs, and pigs. The earliest changes in the alveolar and atrial lining cells were increase in cell size and nuclear hypertrophy, often with clumping of chromatin, and the appearance of one or more large nucleoli. Lack of uniformity of this change from one alveolus to another was characteristic of this reaction. The appearance of a hyaline membrane was infrequent in animals, and this structure will be discussed more fully in the section on the pathology of the irradiation reaction of the human lung. The alveolar walls were affected by an irregularly distributed edema and congestion. Increased cellularity and, if the reaction were more pronounced, thickening and splitting of the elastic fibers were found. More severe changes of fibrosis and hyalinization were not seen because only relatively small doses of irradiation were administered. Bronchial epithelium

showed only an increased number of mucous cells a
ciliated cells to cuboid nonciliated forms. Bronch.
alveolar cells in sensitivity, and similar anaplastic c
smooth muscle, and connective tissue showed no clea
lymphatics, supporting connective tissue septa, an
changes, consisting of edema and congestion earl
fibrosis in the later stages.

Quantities of irradiation well within the thera
actions which must unquestionably severely distu
increase the susceptibility of the lung to secondar

THE CLINICAL PICTURE OF RADIATION DAM

Roentgen irradiation of the human chest is al
the treatment of a malignant neoplasm. Such neop
e.g., mediastinal lymphomas, eosphageal carcinom
carcinoma, but the commonest indication for roent
is carcinoma of the breast. A discussion of the pl
the treatment of breast cancer is outside the scope o
feel qualified to discuss methods of administration o
regardless of the method used, a certain amount of
the underlying lung, and often the effects of pulmon
clinically significant. The frequency with which this
stad[8] encountered lung changes in 5.4 per cent of 3
roentgen therapy for breast cancer, and in 20 per cen
for carcinoma of the esophagus. These figures are
Warren and Spencer,[9] who demonstrated histologic
12 per cent of 234 patients treated for a variety of c

The group of patients who receive roentgen
should be especially emphasized, not only because
single group, but also because radiation in these c
one hemithorax and because their chances for long-te
those of most patients with intrathoracic lesions who
Characteristically, the clinical history is that a mid
with a recent radical mastectomy is given postop
While the patient is still receiving therapy or three
moderately productive cough begins. If the dose
(less than 130 to 140 per cent of the skin unit dos
Leucutia[10]) the cough may persist for several months
If greater quantities of irradiation reached the lun
progressively worse. In some instances the cough is
months have passed. The quantities of sputum produ
a very common symptom, and may be paroxysmal an
a frequent complaint, but hemoptysis is relatively u
patients are in good general health, and this findin
differentiation of the irradiation reaction from pu